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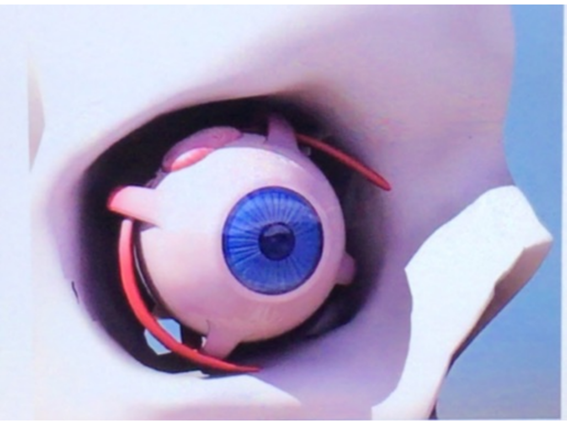


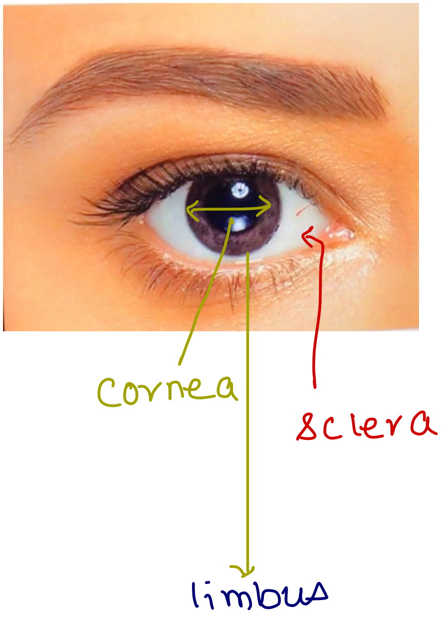
Shape of Eyeball - Aspherical
(Ablate spheroid)

* Vol. of Eyeball : 6ml
* Vol. of orbit : 30ml

Structure of Eyeball

- 3 layers

Outer



Middle

Uvea

- Iris
- Ciliary body
- choroid

Inner

- Retina

Cornea

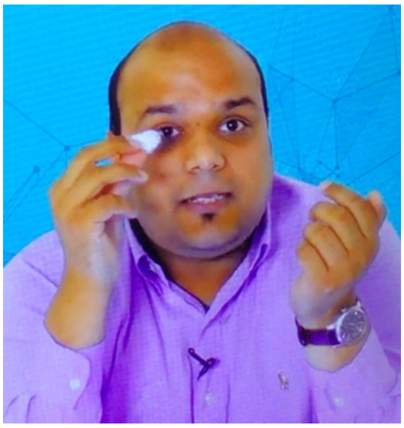
- Blood supply → Avascular
- N/v supply → Trigeminal N/v (opthalmic N/v)

lacrimal, frontal, Nasociliary

Nasociliary ↓ Supplies cornea

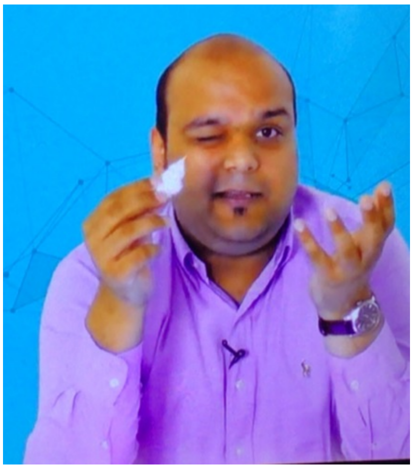
TON

Q) How to test Corneal sensations?



Touch cornea surface c̄ cotton wisp

→ Corneal Blink Reflex



Eye blink
↓
· corneal sensations — Ⓝ

- If not ⇒ Sensations absent or reduced → Corneal Anesthesia

★ H - Herpes
D - Diabetes
L - Leprosy
A - Acoustic Neuroma
C - contact lens
T - Topical Timolol (β blocker - long term)
S - Sx Trauma

Corneal Blink Reflex

Afferent

- CN5

Efferent

- CN7 → Orbicularis Oculi

Note: Bell's palsy

↓

Orbicularis oculi Palsy

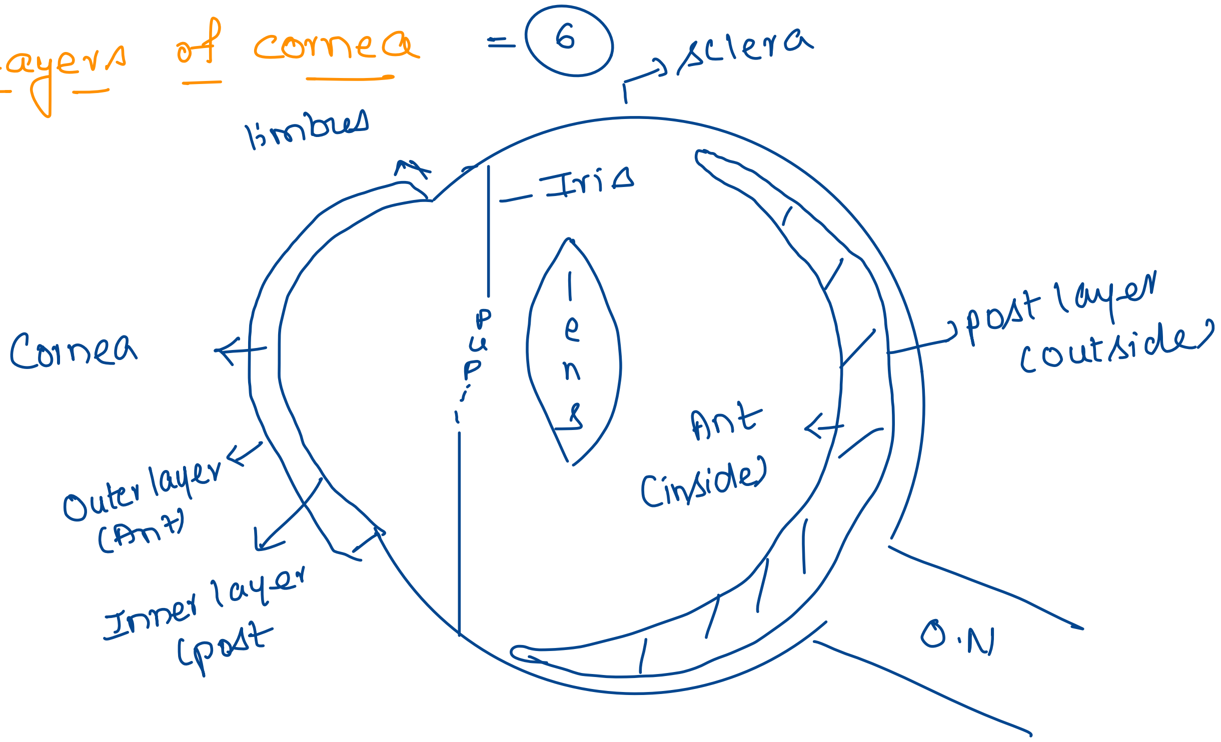
↓

inability to blink

⇓

LAGOPTHALMOS

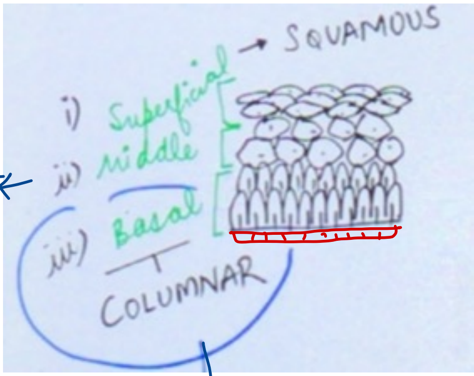
Layers of cornea = ⑥



a) outermost layer → Epithelium

- Non keratinised
- Stratified ←
- Squamous

polygonal ←



↓

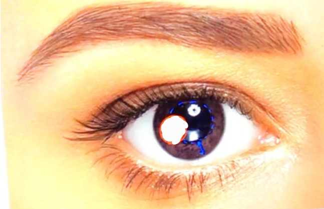
have ability of mitosis ⇒ Corneal epithelium can regenerate

b) Bowman's layer : Not a true basement membrane
↓
PAS +ve

- PAS -ve
- Acellular
↓
Cannot regenerate ⇒ Heals by SCAR
↓
Corneal opacity
↓
Loss of vision

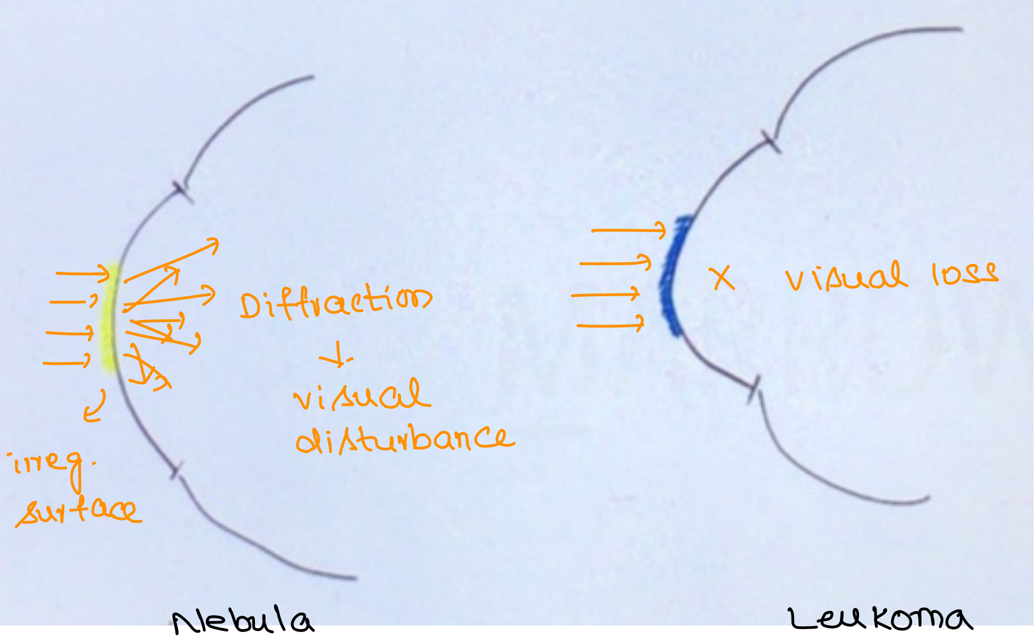
Types of corneal opacity:

	Pupillary margin	Iris structural details
1. Nebula (faint)	V	V
2. Macula	V	NV
3- Leukomatous (dense) complete white)	NV	NV



b) Which type of corneal opacity causes max. visual disturbance or distortion ?

— Nebular



c) **Stroma** : 90% of total corneal thickness

m/c type of collagen : Type 1

m/c type of GAG : Keratin sulphate

d) **Dua's layer** : Strongest corneal layer

[t : 7-14 μ]

e) **Descemet's membrane** : Secreted continuously by underlying endothelial cells

↓

i.e can Regenerate

⇒ Thickness - ↑ throughout life

⇒ Peripheral termination

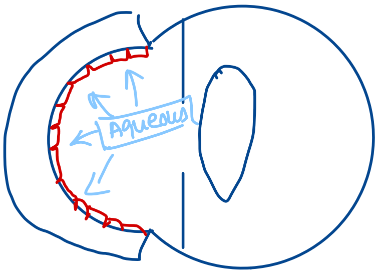
→ Schwalbe's line

f) Inner most / post ⇒ **Endothelium** : Single cell layer

metabolically active layer

Helps to maintain corneal transparency

- Active Na⁺/K⁺ ATPase pump
  ↓
  maintain corneal dehydration
- Tight Jn s b/w adj. endothelial cells



Breech in Endothelium
↓
Edema of Cornea
↓
loss of Transparency

Q) Factors which help to maintain Corneal transparency

→ Tear film

⇒ Corneal avascularity

⇒ Collagen fibres in stroma
- uniform 'd'
- Equal distance b/w all collagen fibrils (< ½ wavelength of light)

⇒ Corneal dehydration by Endothelium

⇒ Ⓝ IOP (↑ IOP → breaks tight Jn's)

⇒ Crystallin proteins in corneal stroma reduce back scatter of light

Endothelial cell count

- 2400 - 3000 cells/mm^2 - Ⓝ

- < 2400 but > 500/mm^2
↓
Cornea adopts by

Polymegathism
- ↑ size

Polymorphism
- change shape

↓
plug the gap

- < 500/mm^2
→ Corneal decompensation
↓
Hazy, edematous Cornea

Q) Which Investigation for Endothelial cell count?

- Specular microscopy

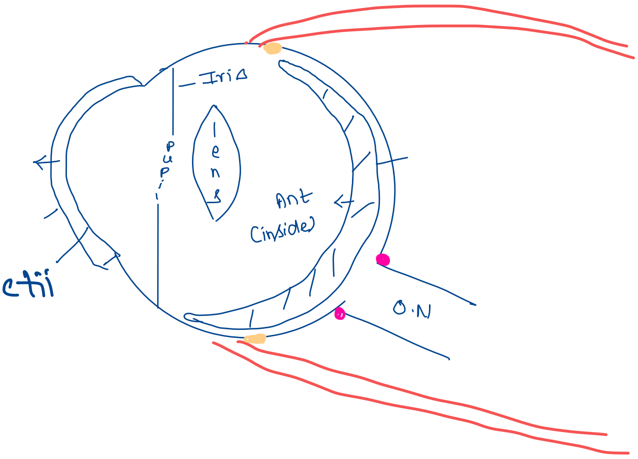
Sclera

- white colour
- posterior part of outermost layer

Thickness

a. sclera - thickest : Around optic N/v

b. sclera thinnest : posterior to insertion of recti



Layers of Sclera

a) Episclera → Episcleritis : Reddish hue

b) Sclera proper → scleritis : violaceous hue

c) Lamina Fusca - innermost layer

Note

Superf. v/s : Reddish

Deep v/s : Bluish

Note

Episcleritis v/s Scleritis : vasoconstrictor test

- No tenderness
⇒ Idiopathic

- Tenderness ⊕
⇒ c̄ Systemic d/s
↓
Rheum. Arthritis

vasoconstrictor test
↓
2.5% phenylephrine
↓

Redness disappear (Blanching)
↓
Episcleritis (superf. v/s)

Doesnot blanch
↓
scleritis

Openings in sclera

Lamina cribrosa : Sieve like opening : allowing ON to pass

- For vortex veins
- For Ant. ciliary Artery, vein, N/vs

Limbus

- Corneoscleral Jn

· Contain stem cells (pluripotent cells)

↓

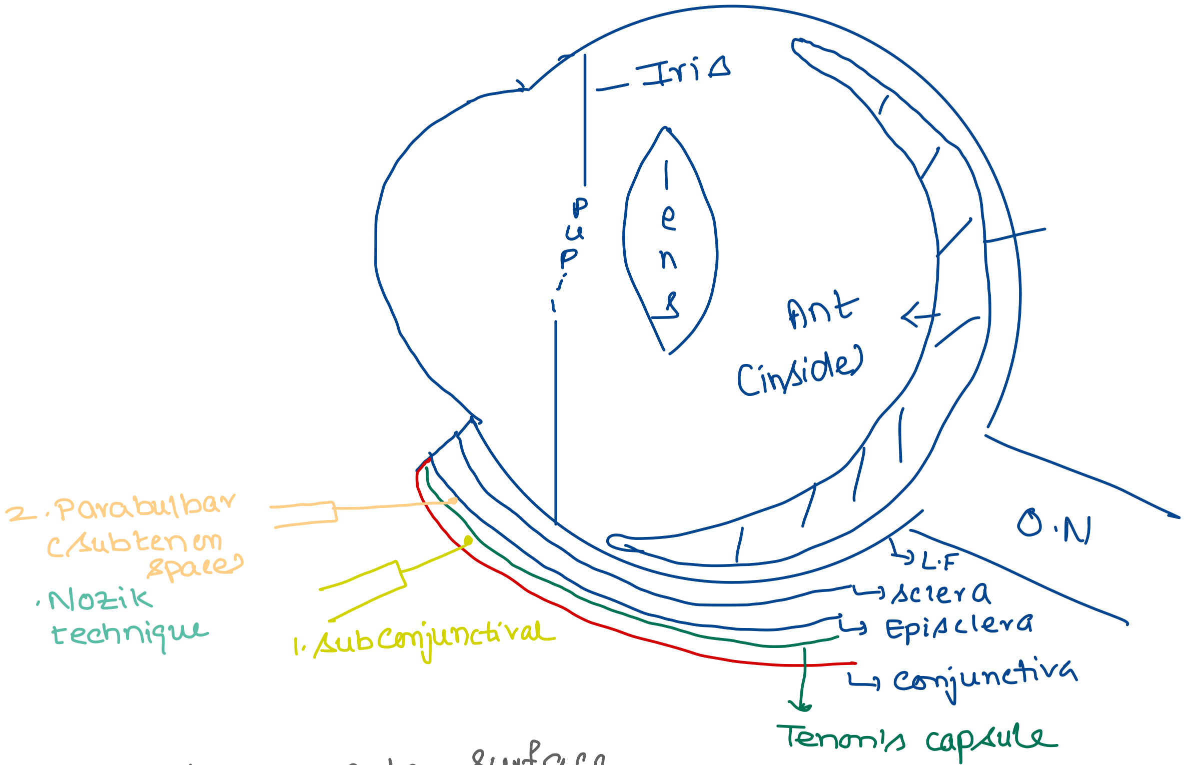
· ability to differentiate
- corneal cells
- Conj cells
- scleral cells

· Location : palisades of vogt

· Universal marker of stem cells : CD34

AIIMS ☆ · Specific marker for limbal stem cells : ABCG2

Routes of ocular drug Administration :

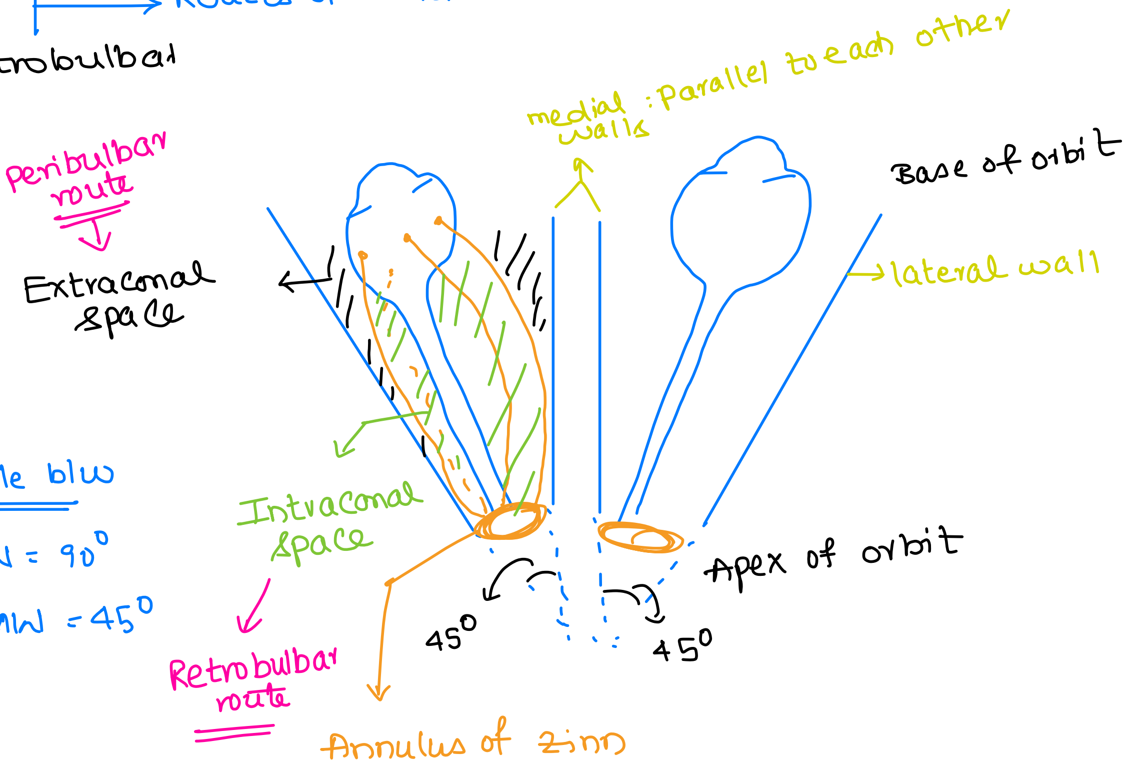


3. Topical : on ocular surface
- Eye drop → admin: atleast 3min b4 eye ointment
- Eye ointment : more time of contact

4. Intravitreal injection

5. Intracameral : Ant· chamber

6. Peribulbar ⟶ Routes of ocular LA.
7. Retrobulbar



* Angle b/w

2 LW = 90°

LW, MW = 45°

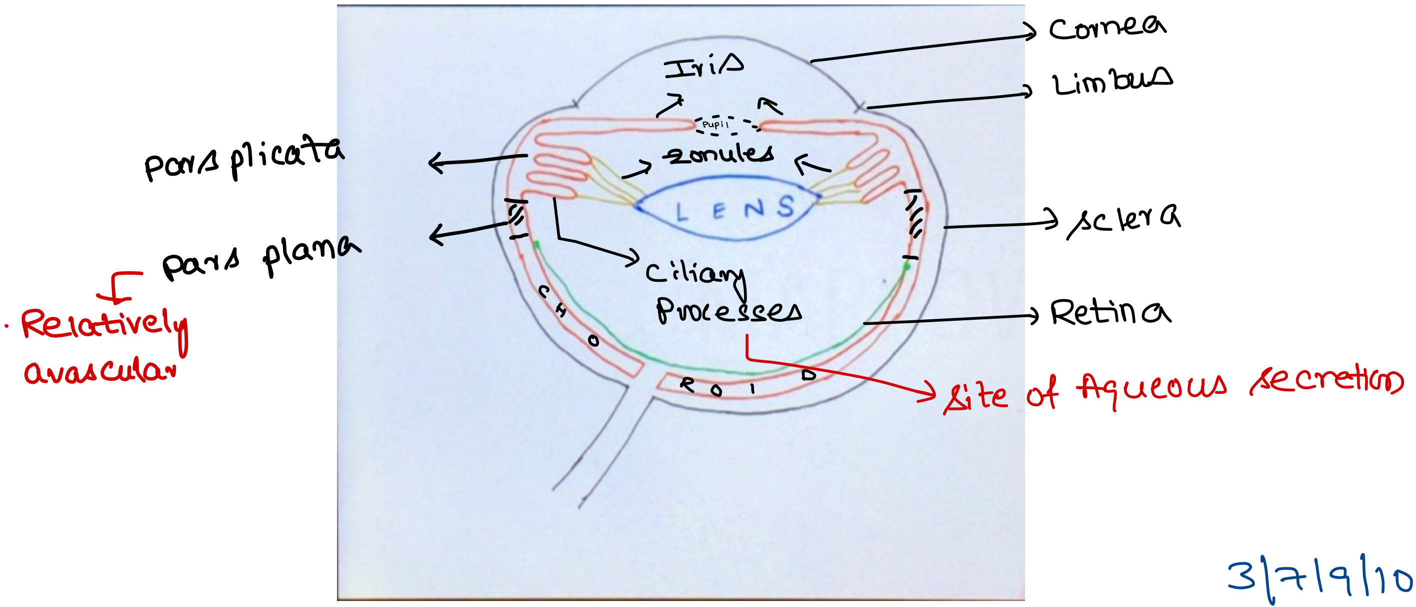
Annulus of Zinn

- Common tendinous ring
- Origin of all EOM (exc: IO)

Middle layer : Uvea

: vascular layer

1. Iris → Ant Uvea
2. ciliary body
   - Pars plicata → Ant Uvea
   - Pars plana. → Intermediate uvea
3. choroid → posterior uvea



3|7|9|10
↓
Parasymp

Iris :

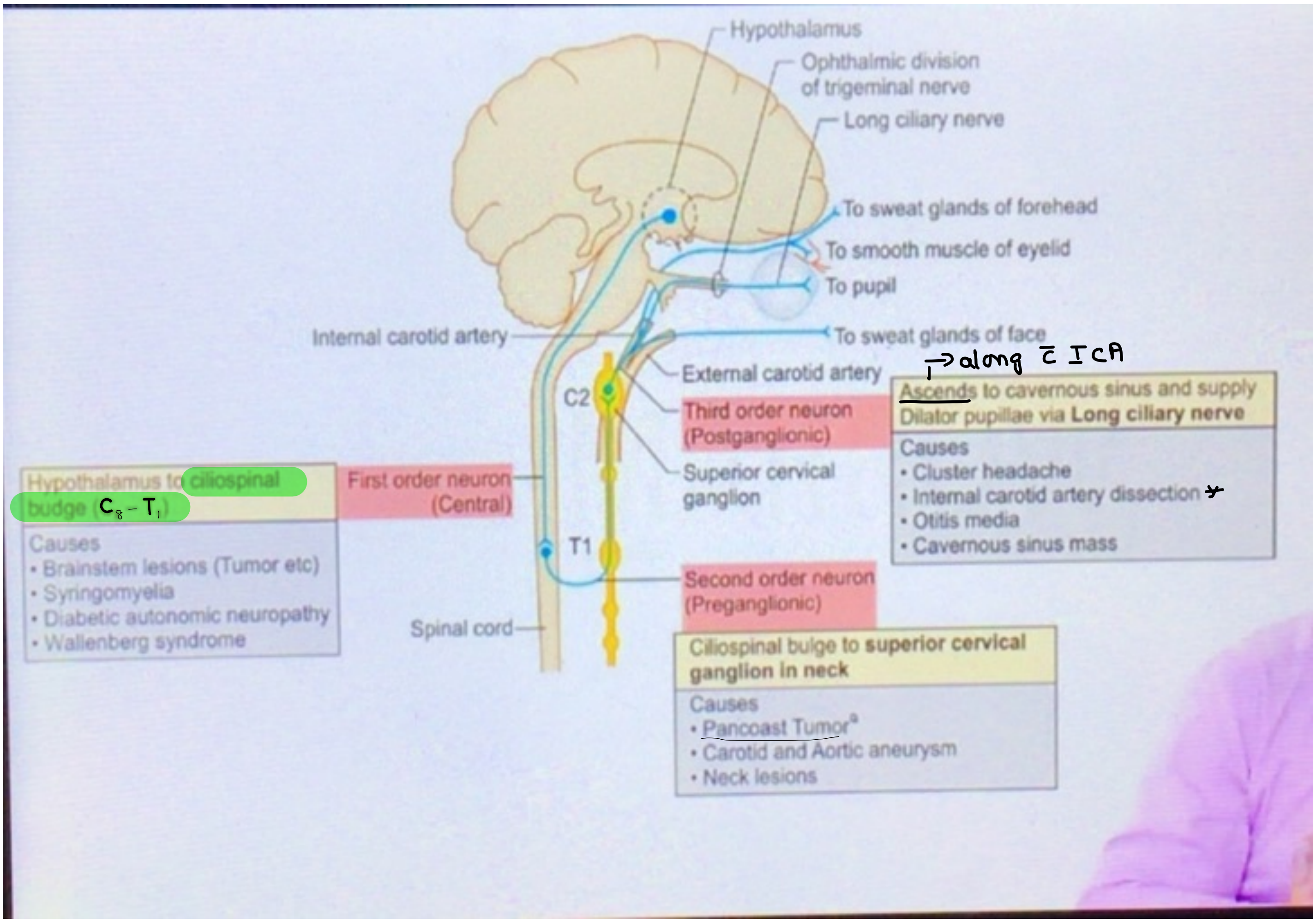
Spinchter pupillae (circular)		Dilator pupillae (Radial)
· miosis	fn	· mydriasis
Parasympathetic ↓ Occulomotor N/v ↓ Inferior branch (also supplies I.O) ↓ short ciliary N/v	N/v Supply	Sympathetic ↓ long ciliary N/v

Note :

· Oculosympathetic palsy = Horner's syndrome

· Oculomotor N/v (parasymp) –
  - Sup branch → LPS
  - Inf branch → sp. pupillae

Sympathetic pathway



Hypothalamus to ciliospinal budge C_8-T_1

First order neuron (Central)

Causes
- Brainstem lesions (Tumor etc)
- Syringomyelia
- Diabetic autonomic neuropathy
- Wallenberg syndrome

Second order neuron (Preganglionic)

Ciliospinal bulge to **superior cervical ganglion in neck**

Causes
- Pancoast Tumor[a]
- Carotid and Aortic aneurysm
- Neck lesions

Third order neuron (Postganglionic)

→ along c̄ ICA

Ascends to cavernous sinus and supply Dilator pupillae via **Long ciliary nerve**

Causes
- Cluster headache
- Internal carotid artery dissection *
- Otitis media
- Cavernous sinus mass

Horners Sx :

H : Heterochromia Iridis (of both Iris = diff.)

- Hypochromic : lost colour
- Hyperchromic : Gain ''

· only seen in Cong. Horner Sx

· i.e: lighter colour iris ⇒ Abnormal

I : Inferior eyelid elevation (x Inf. Tarsal m/s)

M : Miosis (x Dilator pupillae)

A : Anhydrosis (I/L absence of sweating)

P : Ptosis (drooping - upper eyelid : Muller's m/s palsy (≠ LPS))

Muller's m/s → Symp.

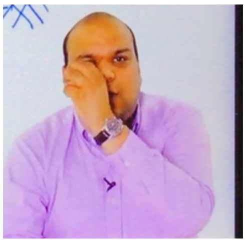
LPS → Parasymp

L : Loss of ciliospinal Reflex

E : Enopthalmos

Sn stimulation

→ cilio spinal Reflex



I/L pupil-dilate

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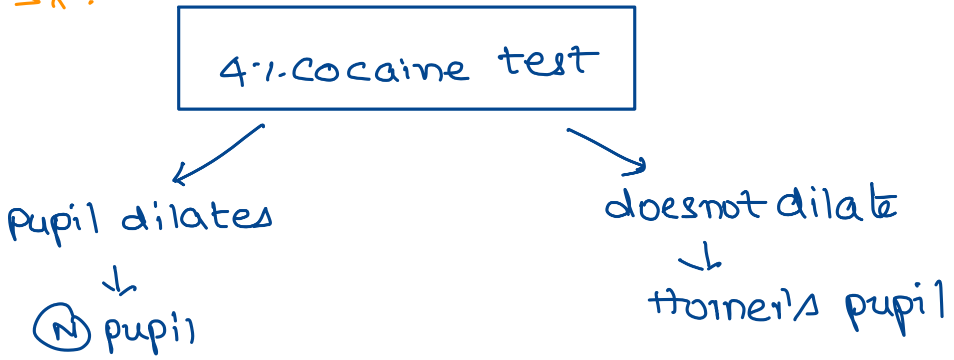
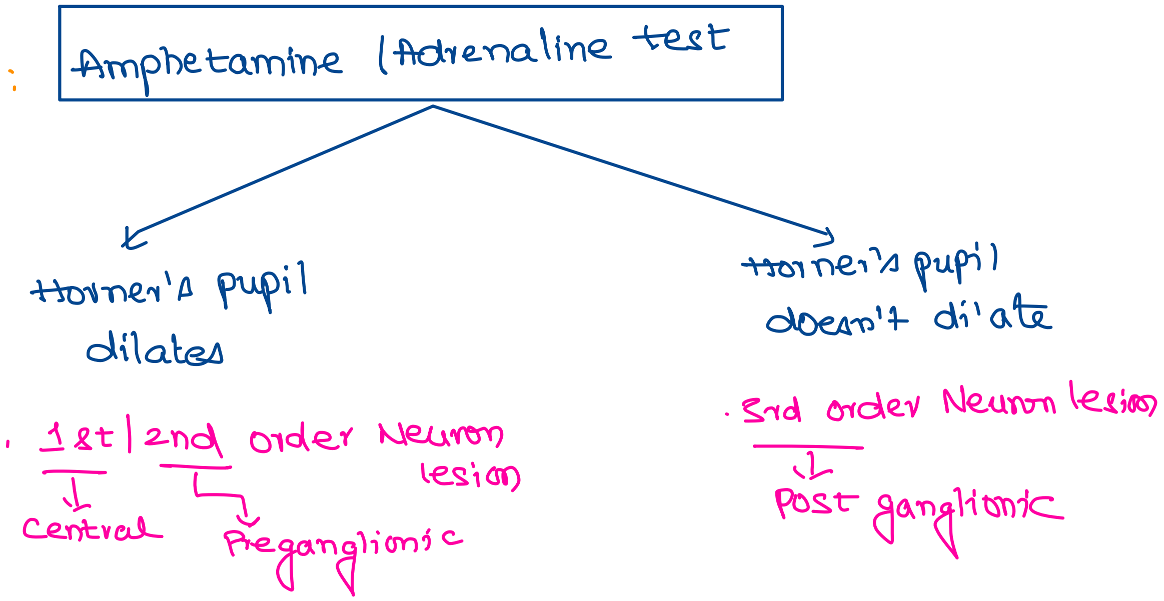
· Dx : Horner Sx :

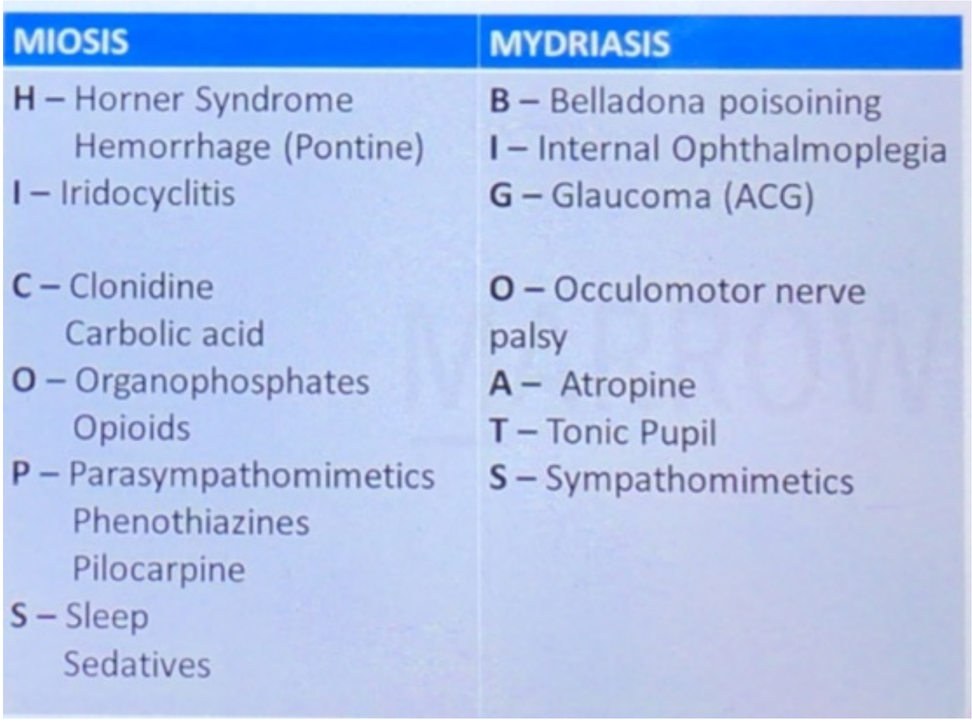


· Site of Lesion :
(localisation)

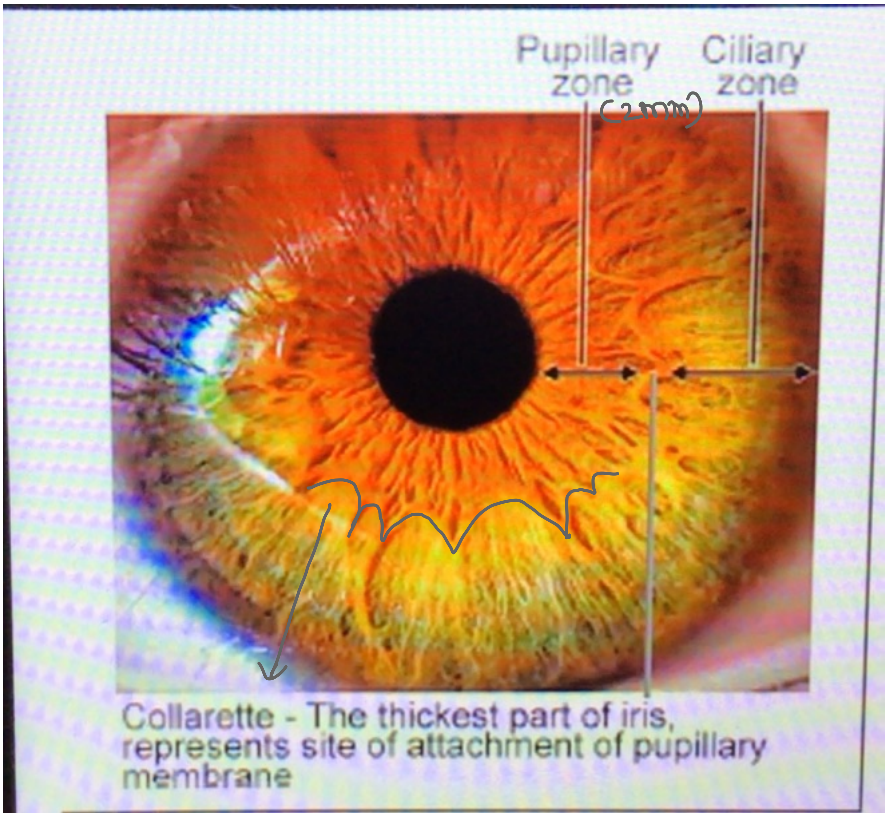

Amphetamine / Adrenaline test
Horner's pupil dilates
· 1st | 2nd order Neuron lesion
Central
Preganglionic
Horner's pupil doesn't dilate
· 3rd order Neuron lesion
Post ganglionic

Note :

Pour de Petit syndrome : Oculosympathetic overactivity



MIOSIS	MYDRIASIS
H – Horner Syndrome Hemorrhage (Pontine)	B – Belladona poisoining
	I – Internal Ophthalmoplegia
I – Iridocyclitis	G – Glaucoma (ACG)
C – Clonidine Carbolic acid	O – Occulomotor nerve palsy
O – Organophosphates Opioids	A – Atropine
	T – Tonic Pupil
P – Parasympathomimetics Phenothiazines Pilocarpine	S – Sympathomimetics
S – Sleep Sedatives	



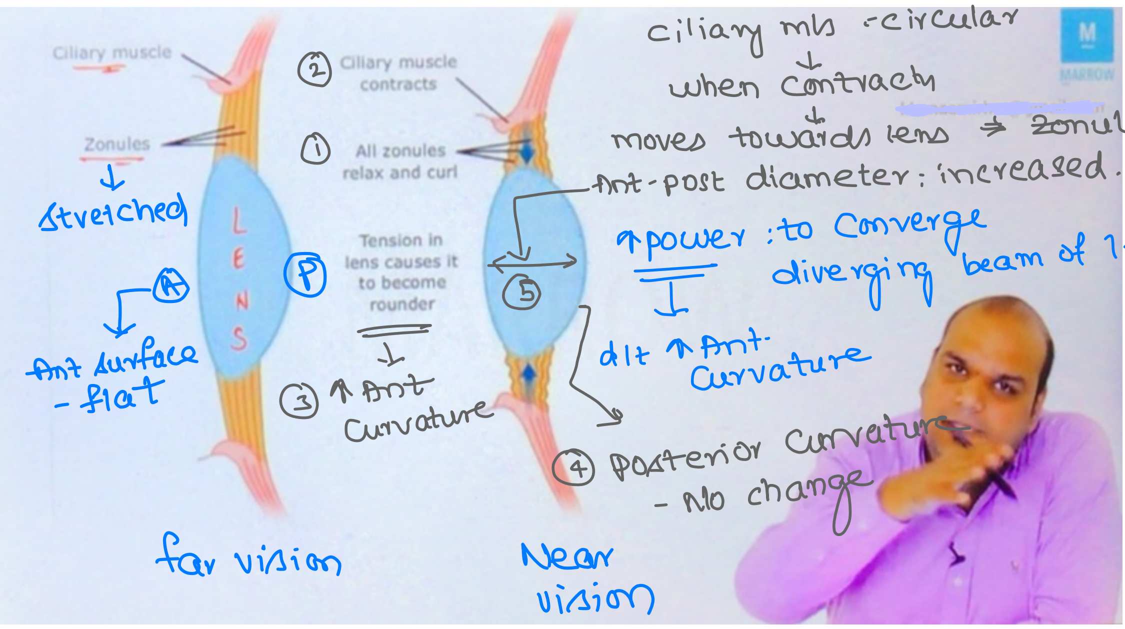
Collarette - The thickest part of iris, represents site of attachment of pupillary membrane

Thickness of Iris:

Thickest	Thinnest
· Collarette	· Root of Iris

ciliary body

- Secretion of Aqueous
- Accomodation : enables near vision
  - part of Near reflex
    - Accomodation
    - Convergence
    - Miosis


Ciliary muscle
Zonules
LENS
② Ciliary muscle contracts
① All zonules relax and curl
Tension in lens causes it to become rounder
⑤
M
MARROW

Zonules
↓
stretched

Ⓐ
↓
Ant surface - flat

Ⓟ

③ ↑ Ant Curvature

ciliary ms - circular
↓
when contracts
↓
moves towards lens → zonules relax
Ant-post diameter: increased.

↑ power : to converge diverging beam of light
↓
d/t ↑ Ant. curvature

④ posterior curvature - No change

far vision

Near vision

Anomolies of Accomodation :

1. Presbyopia : Physiological insufficiency of accomodation
   - Physiological ↓ Age related (~40yrs Age)
   - insufficiency of accomodation ↓ inability
     - to read Newspaper
     - to clearly see mobile

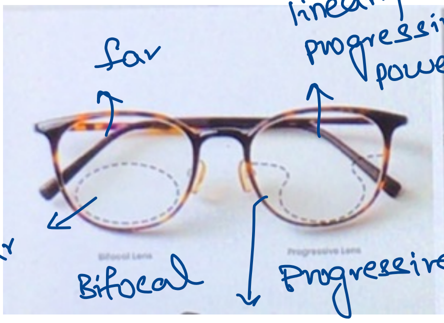
℞ : Spectacles : ⊕ : Convex lens

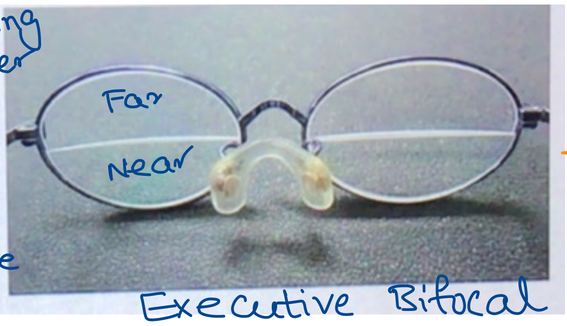
↓

power : depends on age

- 45y : +1.0D to +1.25D
- every 5y → power ↑ by 0.5D
- 50 y : 1.5D to 1.75D
- 55y : +2.0D to +2.25D
- 60y : +2.5D

- pt : Emmetropic → wears monofocal specta
- pt : already using spectacles for any Refractory error : a. Bifocal spects
  - D-shaped
  - Executive
- b. progressive spects ↓ mimics Natural vision (Near → far)





Executive Bifocal ☆ AIIMS

- Now a days used : pediatric Aphakia

2 - Spasm of Accomodation : ciliary m/s : permanently contracted.

↓

Focus @ Near object

↓

Problems in far vision

↓

mimics myopia

[Psuedomyopia]

℞ : Cycloplegia : Temporary pharmocological paralysis of ciliary m/s

↓

Atropine

CHOROID : posterior part of uvea lines the retina

Bruch's membrane : Composed of

a) B.M of RPE

b) Inner collagenous layer

c) Elastic t/s layer

d) outer collagenous layer

e) B.M of choriocapillaries

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Disclaimer

- These Notes do not cover all the points, especially conceptual points, discussed in the videos. The information in this book is meant to complement Marrow videos and should be used in conjunction with the videos. Special emphasis on certain points and MCQ solving approach has to be understood from the videos only.
- Please refer to the slides feature in the Marrow app for all the images discussed in the video. Only select images are included in the Notes.

ANATOMY OF EYE: INNERMOST LAYER AND MISCELLANEOUS

Retina

00:00:11

Retina is the innermost, incomplete layer of the eyeball that covers the eyes only posteriorly.

It is the Neural layer of the eye, consisting of all the nerve fibres that collects to form the optic nerve.

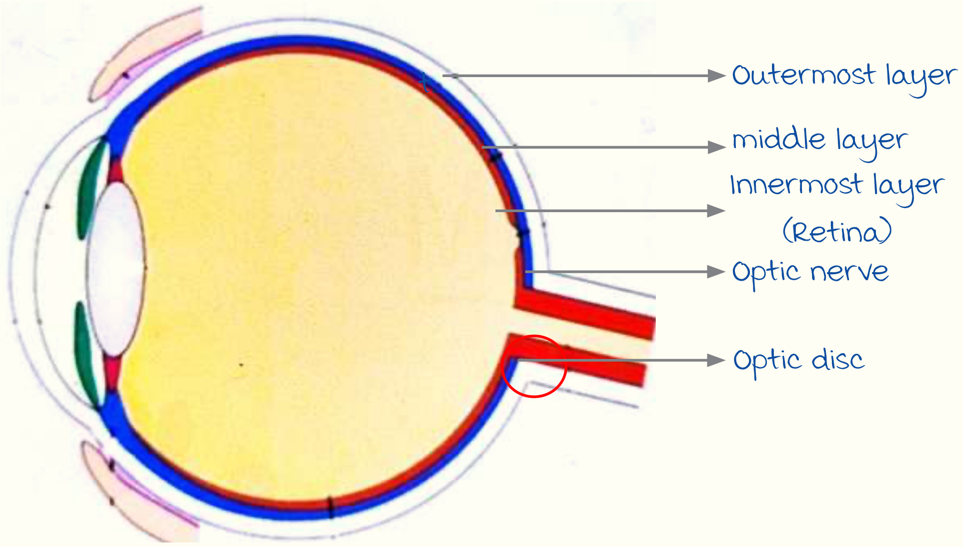
major function is to record the image acting as a film (in the camera) and send it to the cortex via optic nerve.

Gross Anatomy of retina

00:02:10

It has 3 parts :

1. Optic disc / Blind spot :
   - It's the point from where the optic nerve leaves the eyeball or, the Ist part of the optic nerve.
   - It is also known as the blind spot because there are no rods and cones present here, thus no vision.

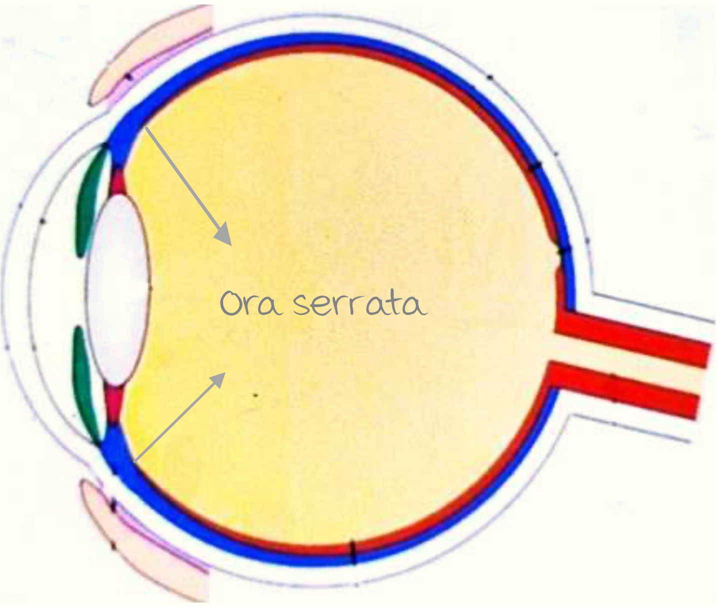


2. Macula :

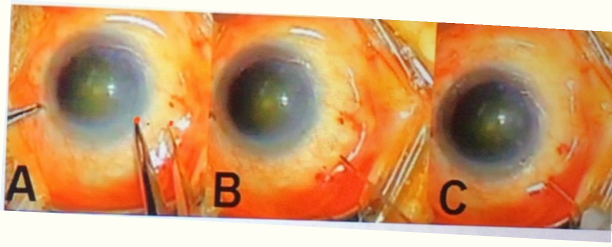
- It is responsible for central vision.
- In all macular diseases, there will be loss of central vision, ex : CME (cystoidmacular edema).
- The centre of macula is called Fovea . Fovea contains maximum concentration of cones, resulting in best image, as it is most sensitive to light.

3. Ora Serrata:

- It's the point of anterior termination of retina, or junction between ciliary body (anterior to ora serrata) and, choroid (posterior to ora serrata).

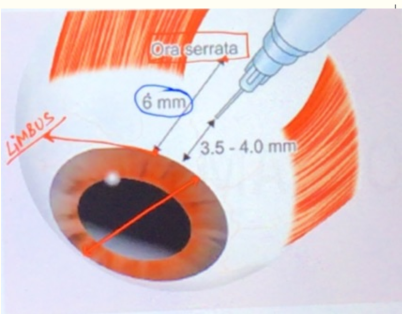


- It is the site for Intra Vitreal injection.
  - Distance between limbus and ora serrata is about 6mm.
  - Appropriate distances for intra vitreal injection, with Limbus as the landmark:
  1. 3mm posterior to limbus – Aphakia.
  2. 3.5 mm posterior to limbus – Pseudophakia.
  3. 4mm posterior to limbus – Phakia.



-Sites for intra vitreal injection:

anterior of ora serrata or,
posterior to limbus or,
between ora serrata and limbus or,
* by piercing sclera and pars plana. *
rel. Avasc-



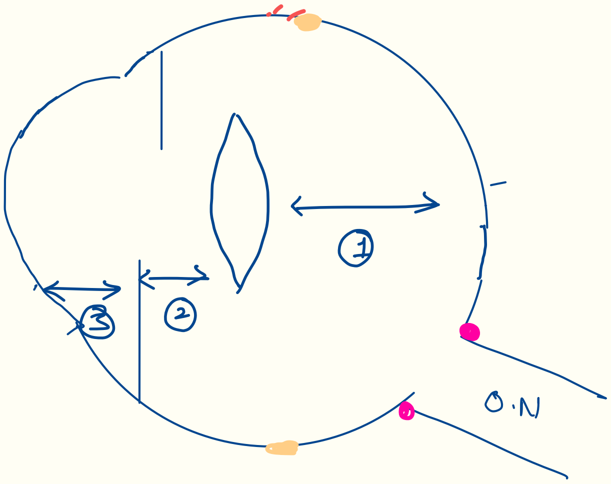
Appropriate site of Intravitreal Inj

- 3mm post to Limbus – Aphakia
- 3.5mm – psuedophakia (IOL)
- 4mm – phakia (Ⓝ crystalline lens)

Active space

Spaces inside the eyeball

00:28:23



Space 1 : posterior segment – contains vitreous
Space 2 : posterior chamber – space b/w iris and lens
Space 3 : anterior chamber – space b/w cornea and iris

Posterior chamber + anterior chamber → anterior segment (contains aqueous)

Aqueous	Vitreous
Liquid state	Gel form (because it contains hyaluronic acid)
Volume is 0.31 ml (0.25ml in anterior chamber and 0.06ml in posterior chamber	Volume is approximately 4ml
Rate of secretion is 2.3microlitre per min. by ciliary processes and, outflow is by trabecular meshwork	Not continuously secreted, as it is formed only during the development of eye

- Facility of aqueous outflow (C-value) – 0.18 to 0.25 microlitre per minute
- ☆ Investigation to measure C-value – Tonography (≠ Tonometry)
- Investigation to measure IOP (exerted by aqueous) –Tonometry

Telegram--@latestpgnotes

- Aqueous contains higher concentrations of:
  - L Lactate
  - A Ascorbate
  - P Pyruvate

  than plasma
- Anterior chamber depth - Normal – 2.5 to 4.4mm

  Increased in young myopic males. : My My

  My ↓ myopes; My ↓ male ↳ young

Blood supply of the eye

00:40:39

Internal carotid artery

↓

Ophthalmic artery

↓ Central retinal artery branches ← travels ē in optic N/v and supplies · Inner 6 layers of retina

↓ posterior ciliary artery

↓ muscular : 2 each from SR, MR, IR & 1 from LR

↓ form Ant. Ciliary A

↓ Pierce sclera 5mm post. to limbus

↓ Supply
- Conjunctiva
- Ant. sclera
- Some part of Iris & C.B.

posterior ciliary artery →

Long posterior ciliary artery (2)

↓

enter eye by piercing sclera, away from O.N

↓

travels b/w sclera & choroid

↓

Reaches upto Ciliary body

↓

Divide into 2 branches

→ supply ciliary m/s

↓

Reach periphery of Iris

↓

Anastomose near Root of Iris

↓

Major Arterial circle of Iris

Short posterior ciliary artery (20)

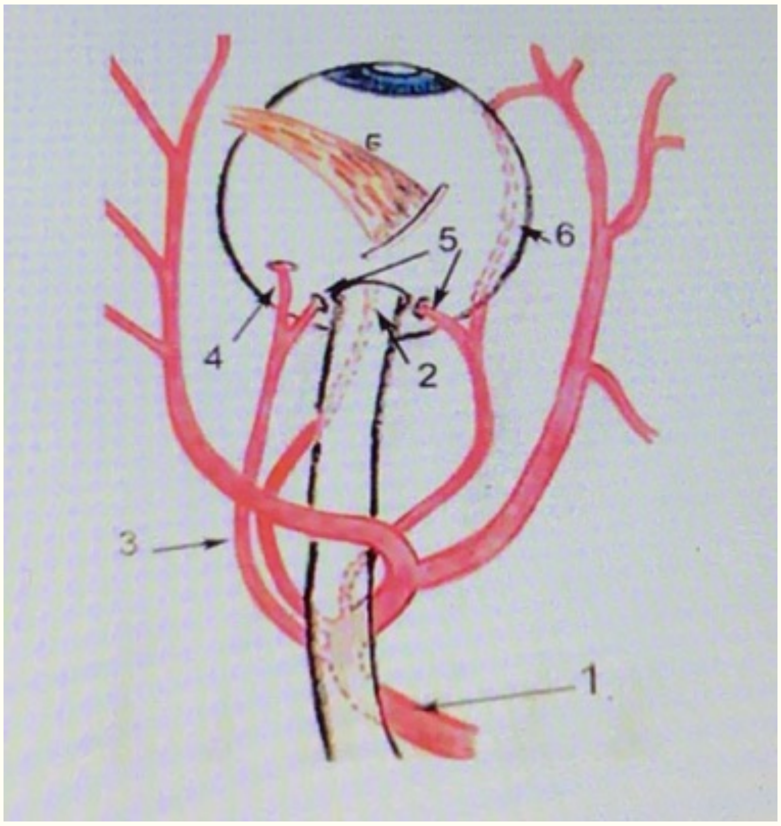
↓
- Sclera
- choroid
- outer 4 layers of retina

Active space

These 2 branches anastomose near the root of iris, to form major arterial circle of iris and supply the periphery of iris.

Branches of major arterial circle move centrally and form minor arterial circle of iris, that supplies the pupillary margin and some central part of iris.

- Short posterior ciliary artery - are 20 in numbers → enters eye by piercing sclera, around the optic nerve.



Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

1. Ophthalmic artery
2. Central retinal artery
3. Posterior ciliary artery
4. Long posterior ciliary artery
5. Short posterior ciliary artery
6. Anterior ciliary artery

Active space

NEURO-OPHTHALMOLOGY: VISUAL FIELD DEFECTS

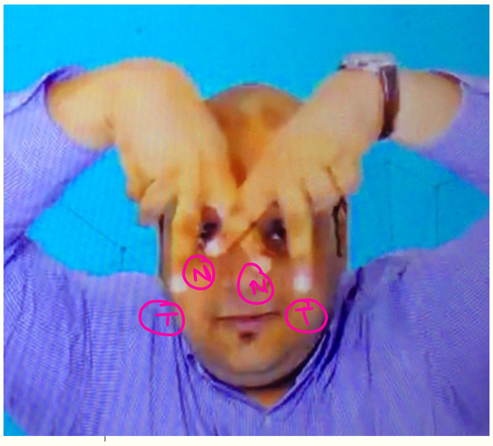
Visual pathway

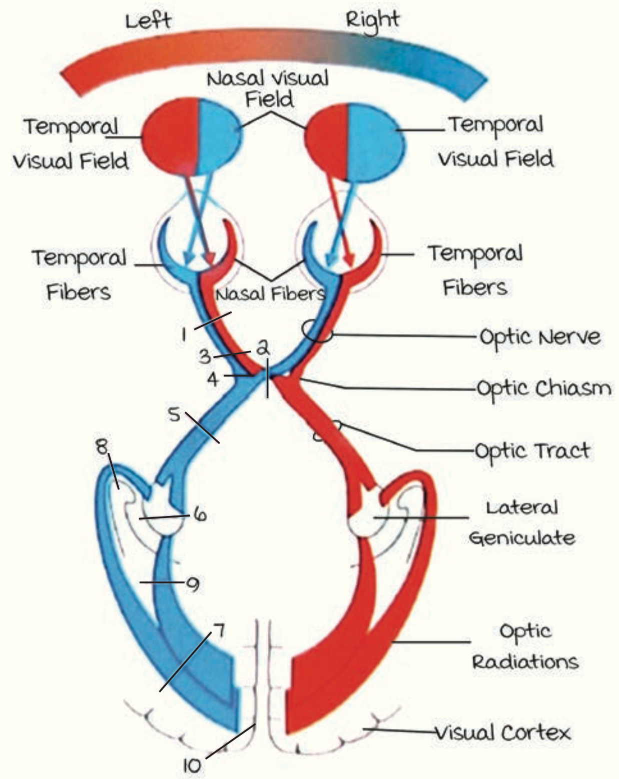
00:03:13

Starts from the retina and goes up to the visual cortex.

Retina :

- Inner part- Nasal
- Outer part- Temporal





: Ⓡ- N, Ⓡ T : I/L T, I/L N

: B/L Ⓝ fibres

: I/L Ⓣ, C/L Ⓝ

" "

" "

: " "

1: Optic Nerve – Contains ipsilateral nasal fibres and the ipsilateral temporal fibres.

2: Optic Chiasma – Contains both side bilateral nasal nerve fibres.

3: Optic Tract – Contains contralateral nasal and Ipsilateral temporal fibres.

4: Lateral Geniculate Body – Contains contralateral nasal and Ipsilateral temporal fibres.

5: Optic radiations – Contains contralateral nasal and Ipsilateral temporal fibres.

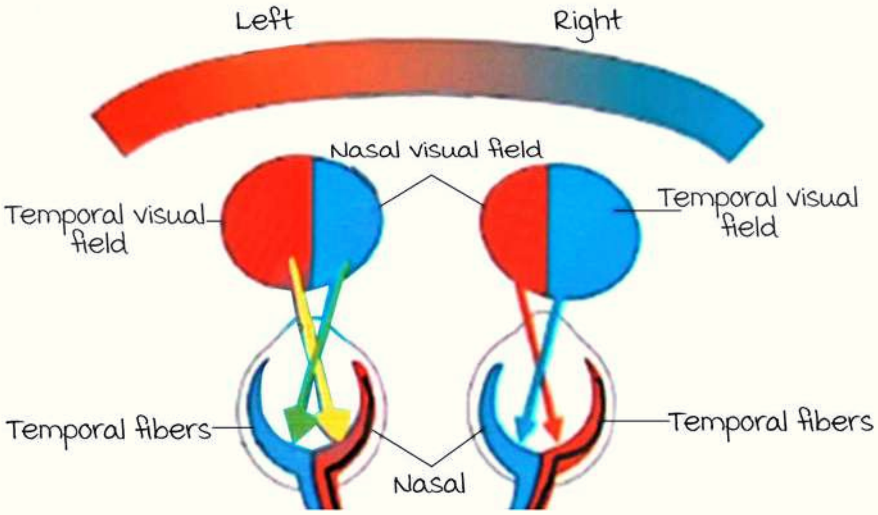
6: Visual Occipital Cortex – Contains contralateral nasal and Ipsilateral temporal fibres.

Correlation of visual pathway with visual field

00:11:50

The outer part is Temporal Visual Field and the inner part is Nasal Visual field.

The whole concept is based on the yellow and green arrows drawn in the diagram given below.



Taking an example of the left eye, if something is visible :

- In the left temporal field then it casts an image on the nasal part of the retina
- In the left nasal field then it casts an image on the temporal part of the retina

Visual Field Coordinates are opposite to the Retinal Fibre coordinates because light travels in a straight line and does not bend.

For example :

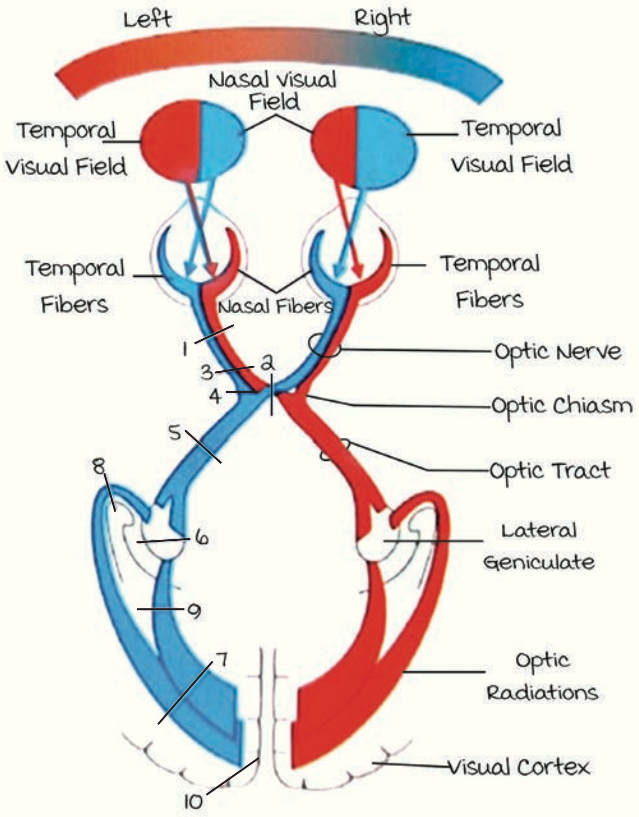
A. Cancer destroys the temporal fibres in both hemispheres : Nasal vision affected.

B. Pituitary adenoma compresses the optic chiasma : Temporal vision affected.

Active space

Visual pathway lesions

00:17:20

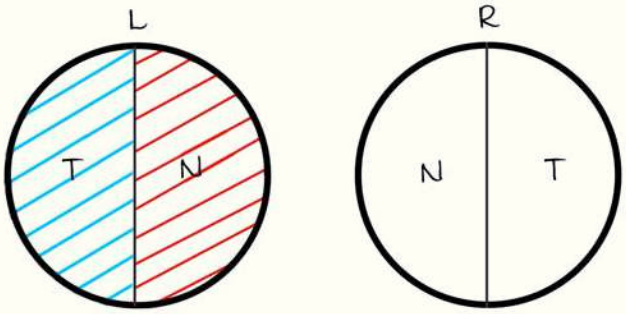


Lesion no 1: lesion in the left optic nerve (ON)

Complete optic nerve lesion

- Temporal Left fibres cut- Nasal visual field gone
- Nasal left fibres cut- Temporal visual field gone

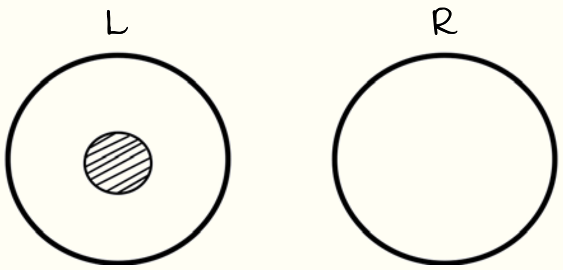
The visual field of the left eye is absent : Left Ipsilateral Anopia



Variant A - Earliest optic nerve lesion

Since most of the diseases pertaining to the optic nerve are progressive in nature, the first part of the optic nerve fibres to be

affected : Macular Fibres (most sensitive to pressure).
Therefore the earliest field loss is the central part : Left Ipsilateral Central Scotoma

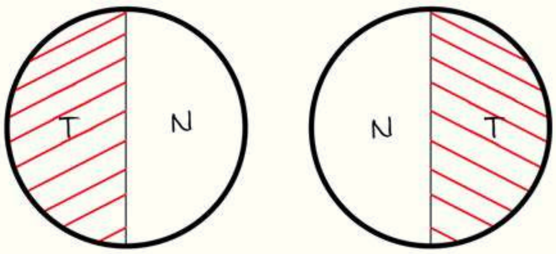


Causes of Central Scotoma : (ON d/s - early)

- Optic Neuritis : m/c → multiple sclerosis
- Heriditary optic Neuropathy
- Toxic optic Neuropathy
- Nutritional optic Neuropathy
- Cone dystrophy : Stargardt's d/s

Lesion no 2: lesion in the central optic chiasma (OC) 00:28:20

Point of crossing so both side nasal fibres are destroyed.
Both side temporal vision affected : Bitemporal Hemianopia (BTHA).
BTHA is also known as Temporal Heteronymous Hemianopia (Heteronymous – from both eyes cannot see towards different sides)



Active space

Causes of BTHA :

3 : 3rd ventricular Glioma
P : Pituitary Adenoma (macro) >10mm : inferior Relation
C – Craniopharyngioma (Rathke's pouch) : superior "
A – Ant. communicating. A Aneurysm
C – Cavernous sinus thrombosis

Buy
↓
BTHA

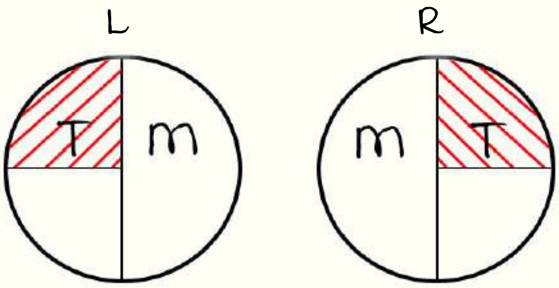
★
'd' b/w optic chiasma & sella turcica
↓
10mm

Variant A – Earliest lesion of Pituitary Adenoma

Only the inferior fibres of the bilateral nasal fibres affected. (as pituitary adenoma is inferior R/2)

So the Superior Temporal part of the visual field is affected :

Bitemporal Superior Quadrantanopia

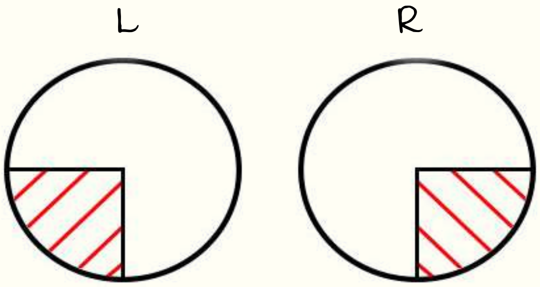


Variant B – Earliest lesion of Craniopharyngioma

Only the superior fibres of the bilateral nasal fibres affected.

So the Inferior Temporal part of the visual field is affected :

Bitemporal Inferior Quadrantanopia



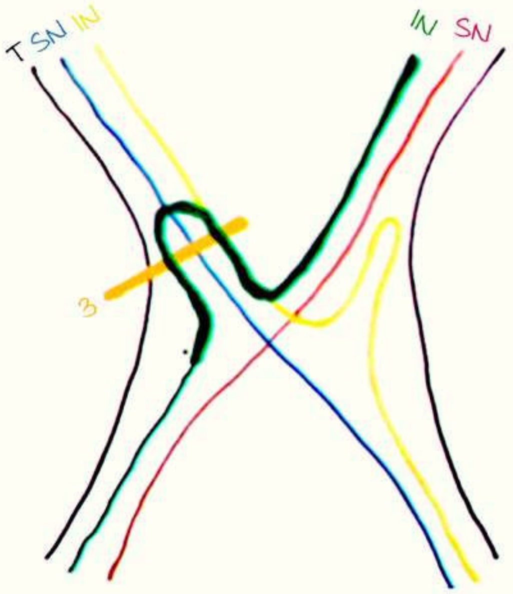
Lesion no 3: lesion in the junction of left optic nerve and optic chiasma

00:44:00

Active space

Also Known as a lesion of "Anterior Knee of Von Willebrand".

The fibres are arranged in a bit complex fashion.



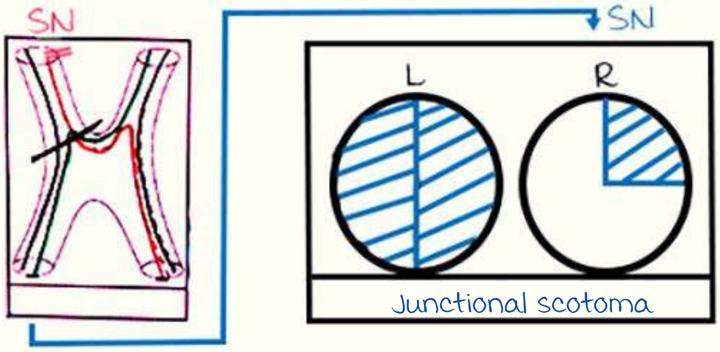
IN – Inferonasal fibres

SN – Superonasal fibres

Fibres affected :

- Left-sided optic nerve fibres: Left eye visual field affected(both nasal and temporal)
- Right-sided inferonasal fibres: Right eye Superotemporal visual field affected

Known as : junctional Scotoma



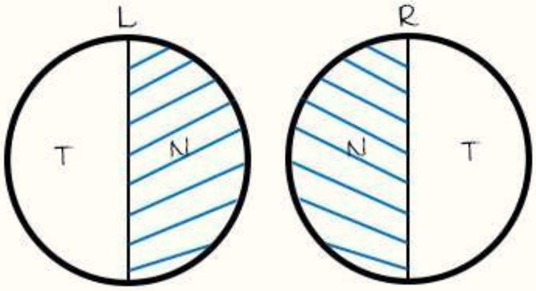
Cause : meningioma *

Lesion no 4: lesion in the lateral part of optic chiasma

00:53:40

* Always a Bilateral lesion. *

⇒ Bilateral temporal nerve fibres are lost, so both eye nasal field lost : Binasal Hemianopia



Causes :

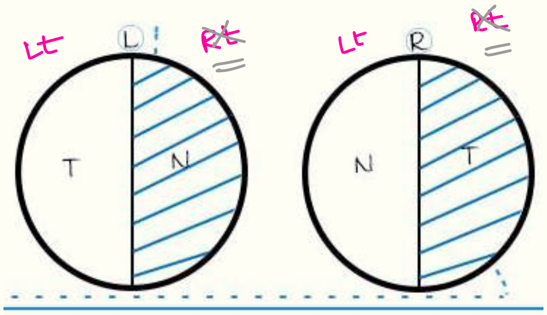
- * Hydrocephalus *
- * Primary empty sella syndrome *

Lesion no 5 : lesion in the left optic tract (OT)

00:57:00

Lesion no 6 : Lesion in the left Lateral Geniculate body (LGB)
Lesion no 7 : Lesion in the full left Optic radiation (OR)

All these have the same fibres, so any lesion at these three points will result in the same visual field defect.
Ipsilateral temporal and contralateral nasal fibres are destroyed.
So, in this case, in the left eye, the nasal visual field is lost and in the right eye, the temporal visual field is lost.
This is a case of Right-sided contralateral Homonymous Hemianopia.
[Homonymous – can't see on the same side in both eyes]
[Hemianopia – half vision is lost in a single eye]



: I cannot see towards Rt side ē both eyes
↓
Rt side

side < Nasal side - for left
Temp side - for Right

Lesion : Lt side → C/L
can't see : Rt side

Active space

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How to differentiate between lesion no 5, 6 and 7

01:03:47

→3rd order

	Optic Tract(5)	Lateral Geniculate Body(6)	Optic radiation (7) ↳4th order
Pupil	ABNORMAL (Wernicke's Pupil Or Behr's Pupil)	Normal	Normal
Optic Atrophy	Bilateral	Bilateral	No atrophy*
Congruity	Incongruous	Incongruous	Congruous

→ After thin Neuron order-changes

How closely visual loss in one eye Relates c̄ visual loss in other eye

Other clinical feature of the Optic Tract Lesion (5) :

→ Passes near to optic tract

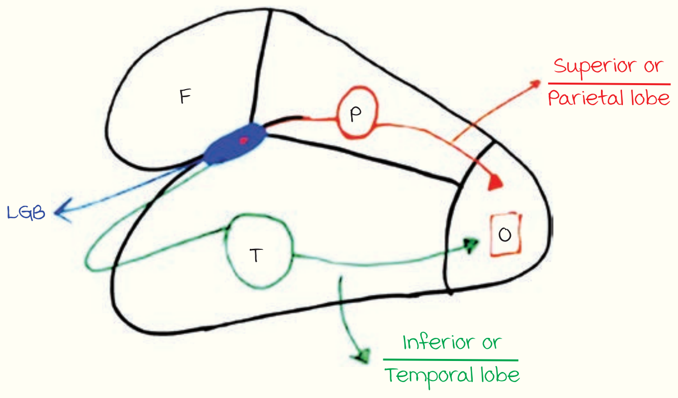
- Contralateral hemiplegia (internal capsule involvement)
- Associated 3rd cranial nerve palsy

Causes : (O.T lesion)

- TB meningitis
- Syphilitic meningitis
- PCA Aneurysm — Crosses Right over optic tract

Lesion no 8 and 9: Lesion in the left optic radiation

01:13:30



Two parts of Optic Radiation (OR) :

- Superior OR- Passes through Parietal Lobe (Baum's Loop) [Lesion no 9]
- Inferior OR- Passes through Temporal Lobe (meyer's Loop) [Lesion no 8]

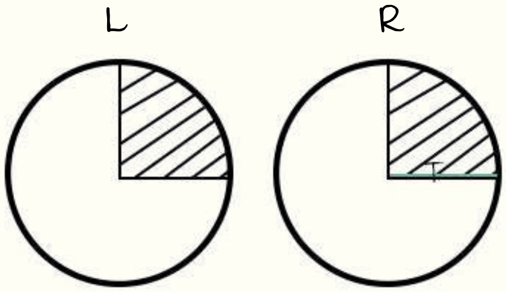
Active space

Lesion 8 : meyer's Loop lesion

Left inferotemporal and right inferonasal fibres lost.

So left - sided superonasal and right - sided superotemporal visual fields are affected.

It is called as : Right-sided contralateral Superior Homonymous Quadrantanopia. Pie in the sky [PITS]

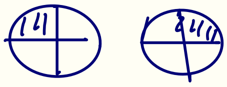


Other features(because of the lesion in the temporal lobe) :

- Uncinate fits
- Seizures ✓
- Hallucinations ✓

Pituitary Adenoma

↓



Heteronymous Quadrantanopia

↓

≠ Pie in sky

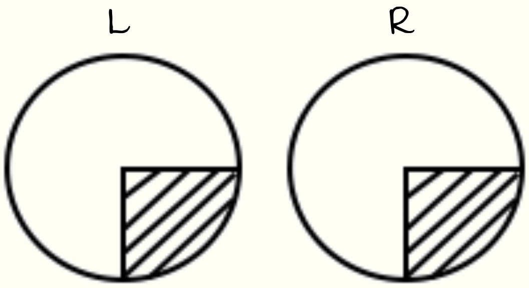
Lesion 9 : Baum's Loop lesion

BF

Left superotemporal and right superonasal fibres lost.

So left-sided inferonasal and right-sided inferotemporal visual fields are affected.

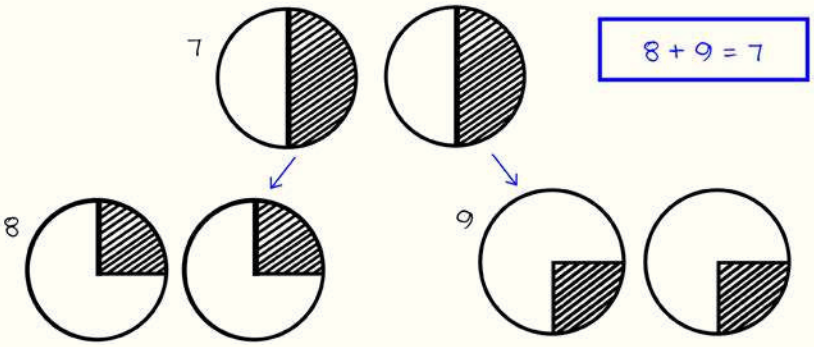
It is called as : Right-sided contralateral Inferior Homonymous Quadrantanopia. "Pie on the floor" [POTF]



Active space

Other features(because of the lesion in the parietal lobe) :

- Acalculia
- Agraphia
- Agnosia
- Apraxia



Lesion no 10: lesion in the visual cortex

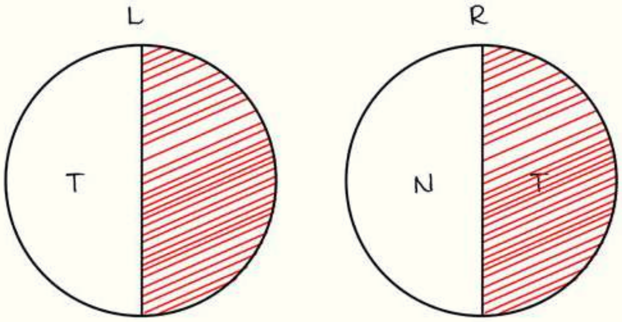
01:30:35

Left temporal and right nasal fibres are destroyed.

So, in this case, in the left eye, the nasal visual field is lost and in the right eye, the temporal visual field is lost

This is a case of Right-sided contralateral Homonymous Hemianopia.

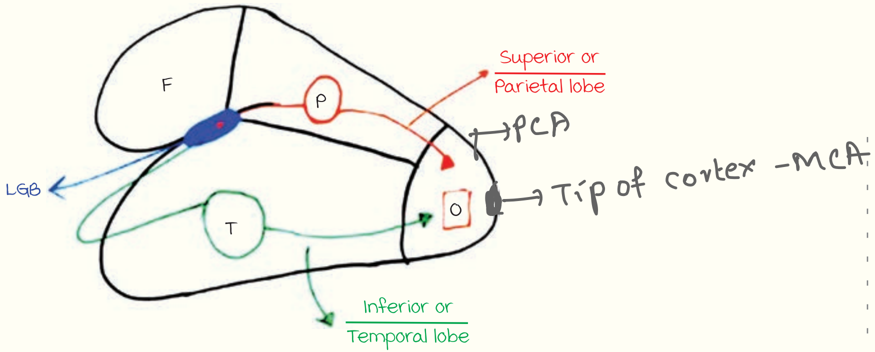
Very rare condition.



Active space

Telegram--@latestpgnotes

Parts :



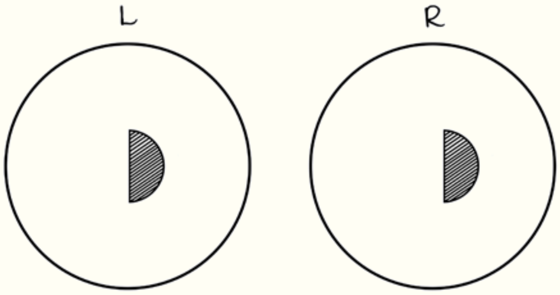
Blood supply : Whole visual cortex is supplied by the Posterior cerebral artery except the tip which is supplied by the middle cerebral artery. Tip represents the macular region

Lesion no 10 a : Tip of the visual cortex (left side)

Left temporal and right nasal fibres are destroyed.

Left nasal side central and the right temporal side central visual field is lost.

This is called the Right-sided contralateral Homonymous macular Defect. Cookie-Cutter Defect

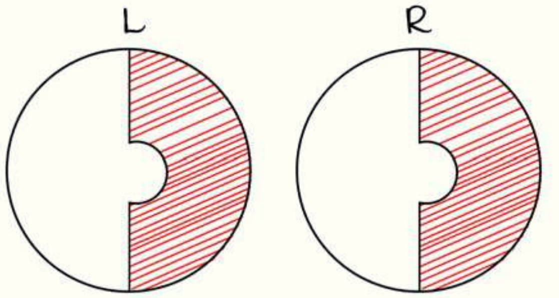


Lesion no 10 b : Visual cortex except Tip (left side)

Left temporal and right nasal fibres are destroyed.

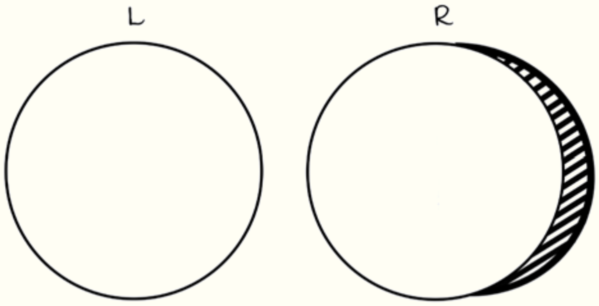
Left nasal side(except macular representation)and the right temporal side(except macular representation) visual field is lost.

This is called as Right-sided contralateral Homonymous Hemianopia with macular Sparing.



Lesion no 10c : Parieto-Occipital fissure(Anteriormost part of the Visual Cortex)(on Left side)

★★ This is called a mono-ocular crescent defect on the contralateral side. ★★



Summary

01:44:10

1. ON - i/l VFD
2. OC - Heteronymous
3. Below OC(OT/LGB/OR) - c/l Homonymous
4. For OR - TI (Temporal Lobe and Inferior OR : PITS) - TI
   PS (Parietal Lobe and Superior OR : POTF) - PS
5. For VC - (Tip=macula)

Miscellaneous topics

01:46:15

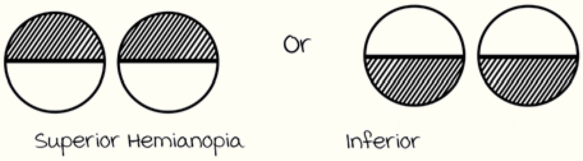
Cogan's Dictum : on OKN test (Optokinetic Nystagmus)

SO EP

Symmetric OKN ↓ Occipital Lobe Lesion (due to trauma)

Erratic Pursuit movement ↓ Parietal Lobe Lesion (due to tumours)

Altitudinal Visual Field defects : Visual field defects which respect the midline.



Causes :

- G – Glaucoma
- O – Optic disc Drusen
- A – AION(Anterior Ischemic optic neuropathy)

AIIMS ★ KeyHole Shape defects : Seen in partial lesions of LGB(due to lesion of Anterior Choroidal artery).

Also known as Quadruple Sectoranopia. *

Cortical Blindness: Lesion in the bilateral visual cortex.

Clinical features :

- Bilateral blind
- → Anton Syndrome : denial of blindness
- Riddoch phenomenon : perception of dim Kinetic but not of a brightly static object.

→ e⁻ activity of visual Cortex

Diagnosis : by test VEP(Visual Evoked Potential)–No recording on VEP.

It can also be used to diagnose Functional Blindness:

- malingering
- Hysteria

Revision

02:03:37

Image 1 : Superior Altitudinal VFD

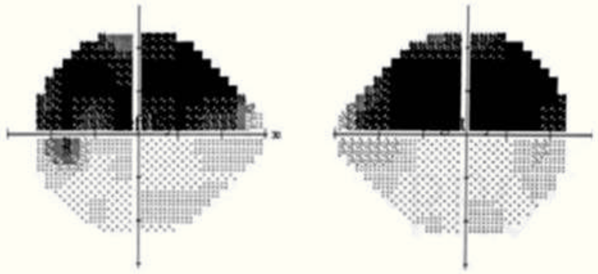


Image 2 : Right – sided meyer's loop lesion

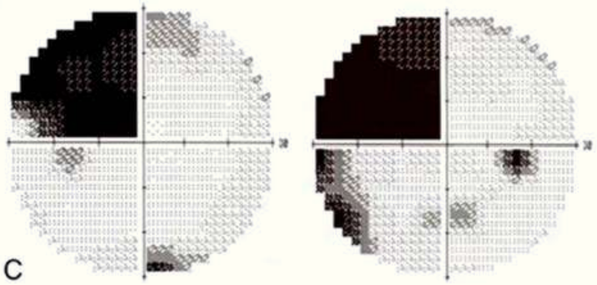
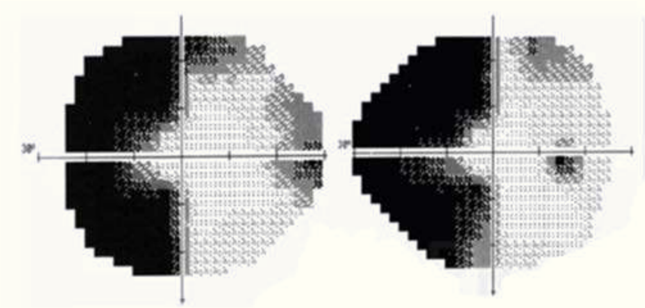


Image 3 : Visual cortex except for the tip



Active space

NEURO-OPHTHALMOLOGY: PUPIL AND ITS LESIONS

Pupillary reflexes

00:00:40

The pupil is under involuntary control.

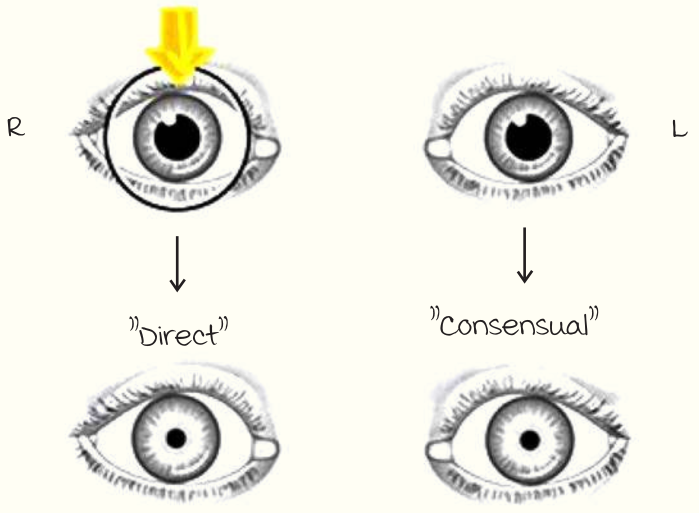
Conditions under which the pupil reflexes :

1. Near Reflex- Accommodation+Convergence+miosis
2. Ciliospinal Reflex-Ipsilateral pupil dilates due to pain on the neck, face, or upper trunk
3. Psychosensory Reflex- dilatation of pupils in response to stress.
4. Light Reflex-

Light Reflex

Direct Light Reflex(DLR)
Constriction of the ipsilateral pupil on which light is throw

Consensual Light Reflex(CLR)
Constriction of the contralateral pupil on which light is NOT thrown

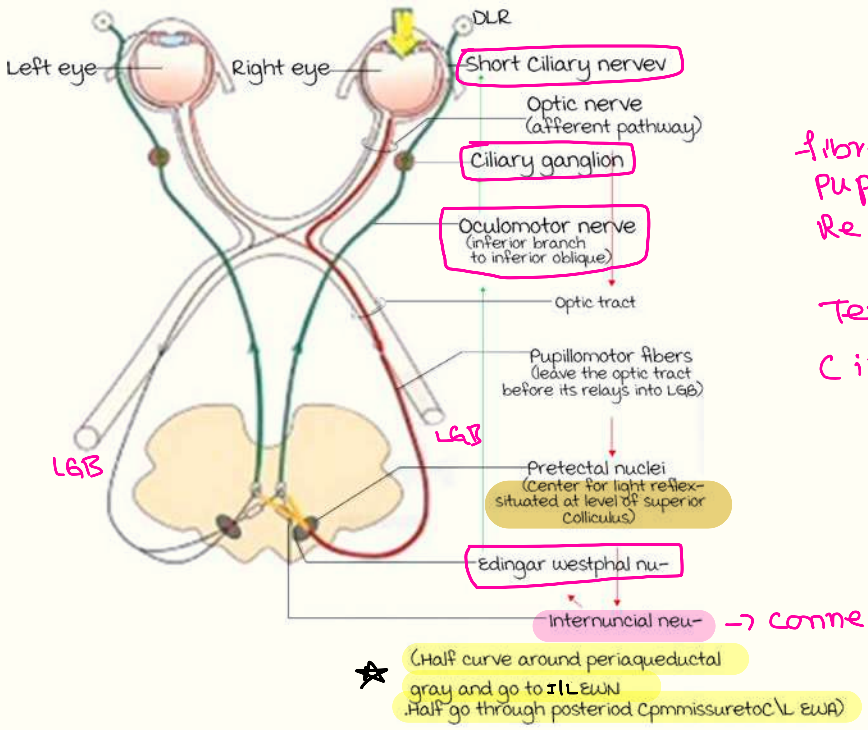


Constriction of stimulated pupil

Constriction of contralateral pupil

Pathway for light reflex

00:09:37


DLR
Left eye
Right eye
Short Ciliary nervev
Optic nerve (afferent pathway)
Ciliary ganglion
Oculomotor nerve (inferior branch to inferior oblique)
Optic tract
Pupillomotor fibers (leave the optic tract before its relays into LGB)
LGB
LGB
Pretectal nuclei (Center for light reflex- situated at level of superior Colliculus)
Edingar westphal nu-
Internuncial neu-
(Half curve around periaqueductal gray and go to I/L EWN
.Half go through posteriod CpmmissuretoC\L EWA)

→ connect pretectal N. & EWN.

fibres for
Pupillary light
Reflex
↓
Temporal
(i.e donot
cross)

Centre for Light Reflex : Pretectal Nuclei(situated at the level of the superior colliculus)

ON
↓
OC
↓
OT
↓
PTN
↓
INN ⟶ EWN (C/L)
↓
(I/L) EWN
↓
3rd N/v
↓
Ciliary Ganglion
↓
Short ciliary N/v'
↓

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Active space

Afferent Nerve : Optic Nerve(CN 2)
Efferent Nerve : Oculomotor Nerve(CN 3)

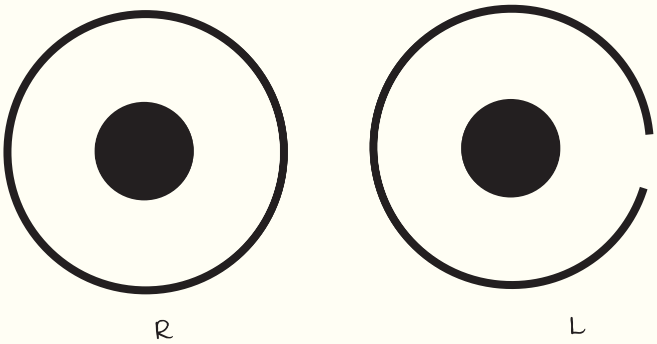
Pupillary abnormalities

00:19:55

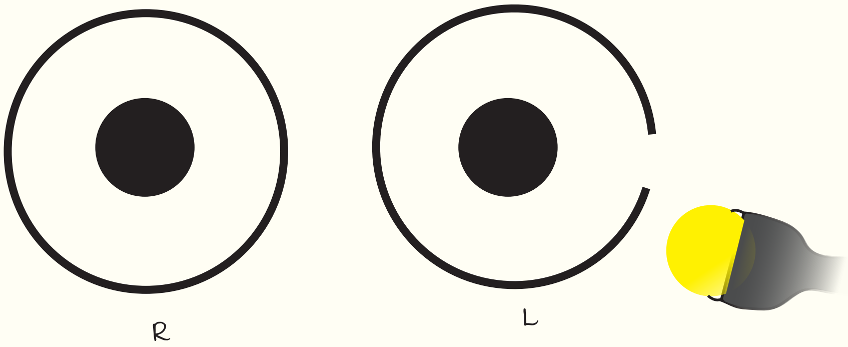
Afferent Pathway Defects(APD)

1. Absolute Afferent Pathway Defect(AAPD)/Total Afferent Pathway Defect(TAPD)/Amaurotic Pupil :

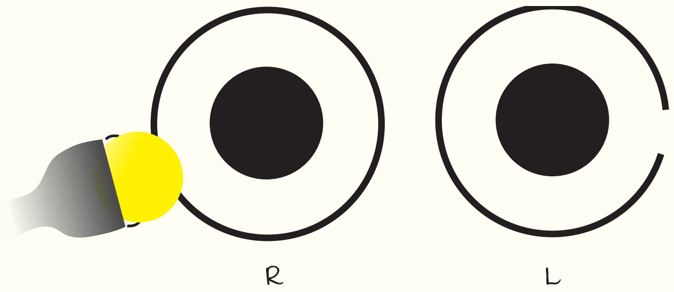
⇒ Caused by complete optic nerve damage.
Assuming that the lesion is in the right optic nerve in the diagrams



a. On presentation : Both Pupil constricted. Both are of equal size - Isocoria.



b. Light on the left side(normal side) : Both Pupil constricted (Isocoria)

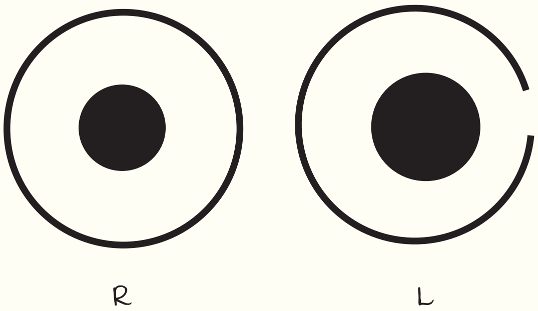


c. Light on the right eye(lesion side): Both Pupils do not react/constrict, remain mid dilated(Isocoria).

In AAPD: Ipsilateral DLR absent
Contralateral CLR absent
+
Ipsilateral Anopia
+
Near reflex is absent?Present

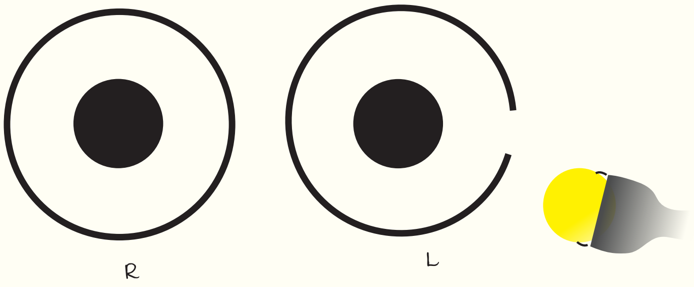
2. Relative Afferent Pathway Defect(RAPD) or Marcus Gunn Pupil: Partial optic nerve lesion
   a. Optic Neuritis
   b. Retinal detachment
   c. Central Retinal Artery Occlusion(CRAO)
   d. Central Retinal Vein Occlusion(CRVO)

Assuming that the lesion is in the right optic nerve in the diagrams.

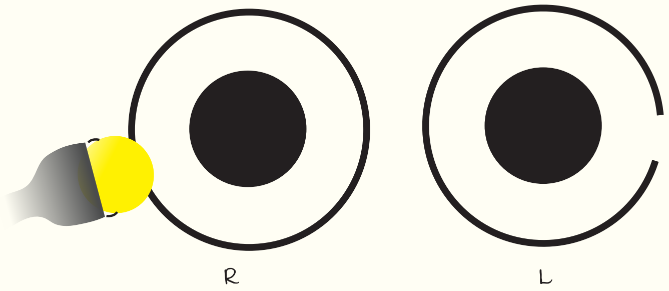


Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

a. On presentation: Both Pupil constricted. Both are of equal size-Isocoria



b. Light on the left side(normal side) : Both Pupil constricted (Isocoria)



c. Light on the right eye (lesion side) : first time : d/t partial lesion ⇒ Pathway still completed ⇓ Isocoria

↓

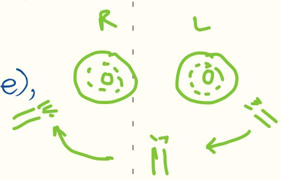
Tested By "Swinging Flashlight Test"

↓

Leads to earlier fatigue of the partially lesioned optic nerve(Right)

↓

So, on withdrawing the light from the normal side(left in this case), As both pupils are in dark, they start to dilate



↓

⇒ On reaching the lesioned side(right eye in this case), due to fatigue The impulse for constriction is weaker than impulse for already occurring dilatation

↓

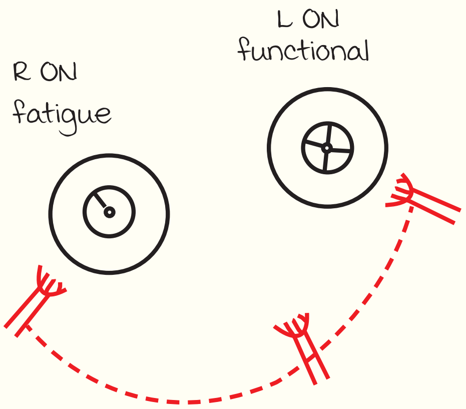
Thus, Both Pupil are seen to dilate when light is thrown on the lesioned side(right eye in this case)

↓

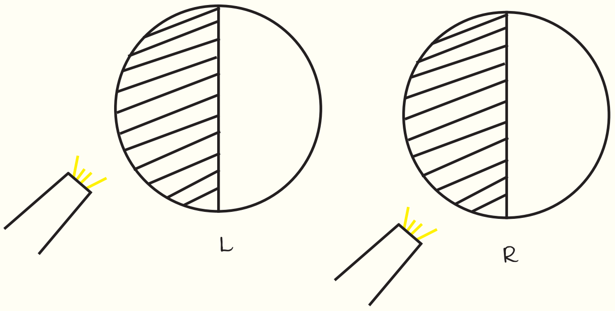
This phenomenon is known as Paradoxical Dilatation/Pupillary Escape Phenomenon

Active space

Telegram--@latestpgnotes



3. Wernicke's Hemianopic pupil
   - Site of lesion : optic tract
   - Light Reflex : Visual Field Defect



a. If the light is thrown in any eye from the side of the visual loss

↓

The pupil does not constrict.

b. If the light is thrown in any eye from the side where the vision is present

↓

Both Pupils are constricted.

Efferent pathway defects (APD)

00:53:05

1. Argyll Robertson Pupil(ARP) :
   - Cause :
     - a. most common- Neurosyphilis(Tertiary syphilis)
     - b. Tabes dorsalis
     - c. Aortic Regurgitation
   - Site :
     - a. Rostral midbrain(aqueductal grey matter)
     - b. Posterior commissure

lesion Affecting internuntial Neurons (BIL)

Active space

Accomodation Reflex Present

Pupillary light Reflex Absent

- Clinical feature :
  a. The pupil does not react to light (LR Absent)
  b. Near reflex is present (NR Present)
  "Light-Near Dissociation"
  Accommodation Reflex Present
  Pupillary Reflex Absent
  c. The pupil doesn't constrict with pilocarpine and dilates poorly with atropine

☆☆ d. The pupil is always seen to be constricted : miosis ☆☆

↓

The lesion in the rostral midbrain also damages the Supranuclear inhibitory fibers to EWN

↓

⇒ EWN keeps on releasing parasympathetic Impulse to sphincter pupillae

↓

Thus pupil remains constricted

"We can say, Pupil don't dilate in Dark"

AIIMS 2020
- B/L constricted pupil donot react in dark? : ARP

Argyll vs Horner syndrome

01:05:30

1. In Horner syndrome :
   - m/c ly unilateral
   - Even though miosis ⊕ d/t dilator pupillae palsy

↓

Show a phenomena : Dilatation lag

↓

In darkness : for 1st 5 sec = doesn't dilate
5-15 sec : starts to dilate
5 min : complete dilatation

2. Tonic Pupil/Adie's Pupil :

Site of lesion : Ciliary Ganglion | short ciliary N/v

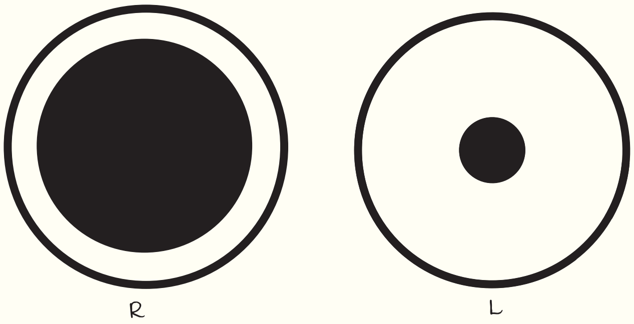
Cause :
1. Orbital Trauma
2. HZV Ganglionitis
3. Diabetes

Active space

4. Alcoholism
5. Holmes-Adie Pupil : Tonic pupil + diminished deep tendon reflexes.

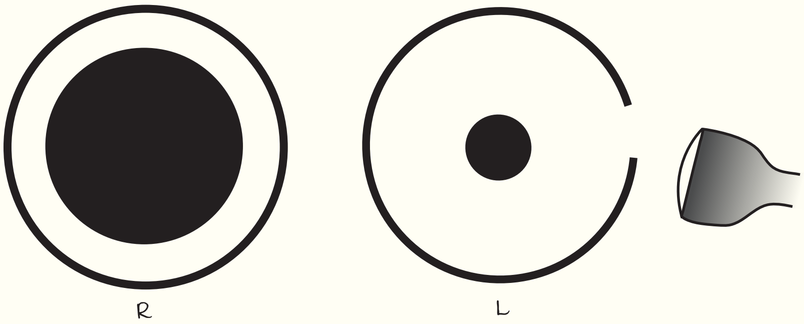
Assuming a lesion in the Right ciliary ganglion

efferent -abn



a. On presentation :

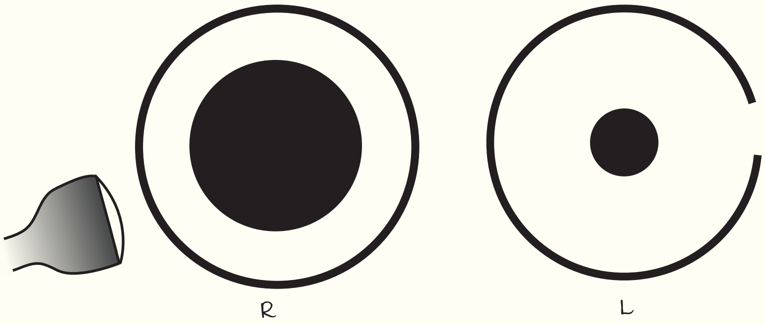
- Right ipsilateral pupil- dilated
- Left contralateral pupil- constricted
- Anisocoria- both pupils of different sizes.



b. Light on the left side(normal side) :

- Right ipsilateral pupil- does not constrict. Ipsilateral Consensual Light Reflex Absent
- Left contralateral pupil – constricts

? → site of lesion : Rt
No RX2 : Rt



c. Light on the right eye(lesion side) :

- Right ipsilateral pupil- does not constrict. Ipsilateral Direct Light Reflex Absent
- Left contralateral pupil- constricts

" Near Reflex is present" - But shows slowly dilating vermiform movements

3. Pharmacological mydriasis :

- Use of Atropine(assume in right eye)

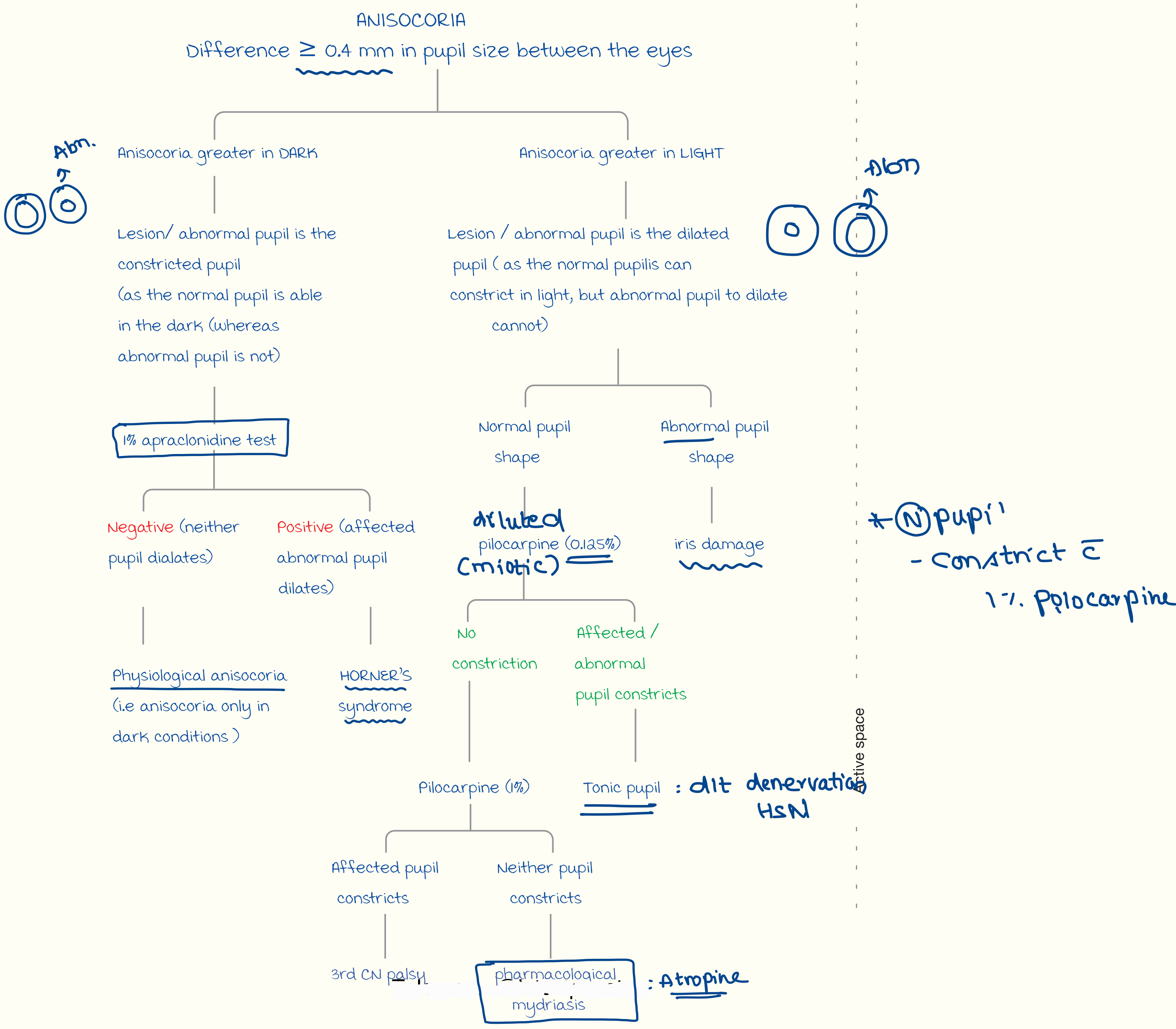
↓

The right pupil is dilated and does not constrict to light

- Tonic Pupil- NR present, LR absent
- Pharmacological mydriasis- NR absent, LR absent

Anisocoria flowchart

01:27:50


ANISOCORIA
Difference ≥ 0.4 mm in pupil size between the eyes
Abn.
Anisocoria greater in DARK
Anisocoria greater in LIGHT
Abn
Lesion/ abnormal pupil is the constricted pupil (as the normal pupil is able in the dark (whereas abnormal pupil is not)
Lesion / abnormal pupil is the dilated pupil (as the normal pupilis can constrict in light, but abnormal pupil to dilate cannot)
1% apraclonidine test
Normal pupil shape
Abnormal pupil shape
Negative (neither pupil dialates)
Positive (affected abnormal pupil dilates)
diluted
pilocarpine (0.125%)
(miotic)
iris damage
+ Ⓝ pupil
- constrict c̄
1%. Pilocarpine
No constriction
Affected / abnormal pupil constricts
Physiological anisocoria (i.e anisocoria only in dark conditions)
HORNER'S syndrome
Pilocarpine (1%)
Tonic pupil
: d/t denervation
HSN
Affected pupil constricts
Neither pupil constricts
3rd CN palsy
pharmacological mydriasis
: Atropine

Active space

- Normal pupil constricts with 1% Pilocarpine.
- If the pupil constricts even with 0.125% Pilocarpine then it is Tonic Pupil, due to denervation Hypersensitivity.

- The pupil does not constrict with 1% Pilocarpine: Pharmacological mydriasis
- The pupil dilates with diluted 1% Pilocarpine : Tonic Pupil

NEURO-OPHTHALMOLOGY: OPTIC ATROPHY, COLOUR BLINDNESS AND MISCELLANEOUS

Optic atrophy 00:00:10

- Optic atrophy is a condition that affects the optic nerve (Atrophy means to waste away or deteriorate).
- Signs :
  1. Pallor of optic disc.
  2. Kestenbaum index – ratio of small to large blood vessels on optic disc surface.
     - normally this ratio = 10
     - but, in optic atrophy = <7
- Optic atrophy doesn't corelate to visual loss. : depends on portion affected
- Types of optic atrophy :

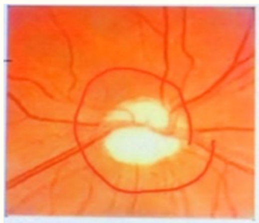
Prev : a/s in O.N

a/s in Retina

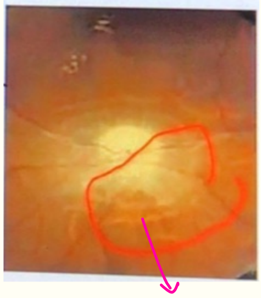
Ⓝ : pink

	Primary optic atrophy	Secondary optic atrophy	Consecutive optic atrophy
Definition	Optic atrophy without any antecedent optic disc swelling	optic atrophy after optic disc swelling	optic atrophy due to diseases of inner retina and its blood supply
Colour of optic disc	Chalky white	Dirty grey white with ill-defined margins	Waxy and pale
Causes	– optic neuritis – tumors – trauma – hereditary diseases of optic nerve – toxic diseases of optic nerve	– papilledema – papillitis – AION	– retinal diseases like CRAO, CRVO, retinitis pigmentosa, etc

1° OA



2° O.A



→ blurring/ill defined margins

Anatomy of optic nerve 00:14:23

- Optic nerve :

  2nd cranial nerve.

  is the collection of axons of ganglion cells. ☆

  cannot regenerate.

Active space

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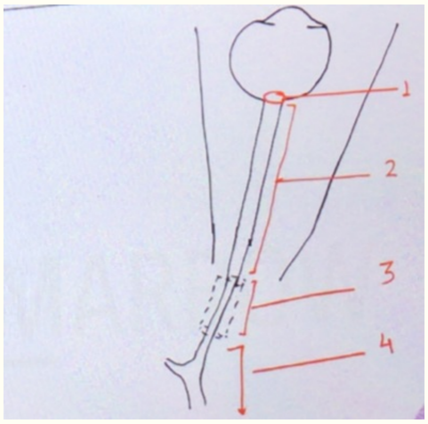


total length is 47 to 50 mm.
is divided into 4 parts.

non myelinated

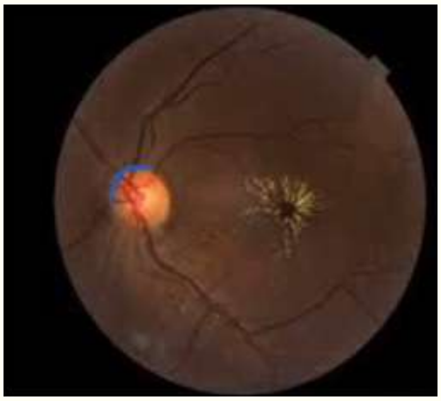
1st Intraocular part	Smallest part; 1mm	Also known as optic disc
2nd Intraorbital part	Largest part; 30mm	Optic nerve acquires myelination; * thus, diameter increases
3rd Intracanalicular part	6–9mm (7.5) x2 x2	Ophthalmic artery crosses over optic nerve
4th Intracranial part	10–15mm	

Optic neuritis

00:22:13

- Optic neuritis is the inflammation of the optic nerve.
- 3 types :

Papillitis	Retrobulbar neuritis	Neuroretinitis
Inflammation of, only optic disc	Inflammn of O.N except O.D	Inflammation of O.D & macula
most common in children	m/c in Adults	
Hyperaemic optic disc and edema seen, due to inflammation	Neither the pt, nor the doctor can see anything	Presents c̄ macular star appearance



d/t deposit of exudates around the macule
↑
macular star appearance

Clinical features:

2x CUP Mar

00:28:53

- Earliest sign – marcus Gunn pupil. (RAPD)
- Central scotoma.
- Colour blindness. (Acq CB : All : Red - Green)
- Unilateral loss of vision.
- Pain (retrobulbar) on ocular movement.

(Also in M.S)

- Uhthoff sign – exacerbation of symptoms due to increased body temperature, eg: exercise.
- Pulfrich phenomenon – is altered perception of kinetic objects.

Treatment: 00:34:38

1. ONTT (optic neuritis treatment trial)	• IV methylprednisolone 1 gm daily for 3 days, followed by Oral prednisolone 1 mg/kg/day for 11 days ☆☆ • This treatment hastens recovery but doesn't affect final visual outcome. • Only oral steroids increases risk of recurrence ($\bar{c}$ out IV loading dose)
2. CHAMPS trial	States beta-interferon 1A reduces the risk of multiple sclerosis after optic neuritis.
3. Glatiramer acetate	20mg, subcutaneously.

IV : 1g/d x 3 d
PO : 1mg/kg/d x 11 d

FIFN1A
↓
⊖
O.N ⇌ M.S

Hereditary optic neuropathy 00:39:45

wolf ne jehr khaya aur
who behri hogayi aur
Leber kharab Hogaya

1. Kjer – autosomal dominant
2. Behr – autosomal recessive; associated with mental retardation, ataxia, nystagmus (MAN)
3. Wolfram – autosomal recessive; also known as DIDMOAD, because of its association with diabetes insipidus, diabetes mellitus, optic atrophy and deafness
3. Leber (LHON-Leber hereditary optic neuropathy) –
⇒ mitochondrial inheritance.

LHON :

- This is a point mutation at codon 11778.
- Maternally transmitted.
- ⇒ More commonly manifested in males.
- Clinical features : – bilateral painless loss of vision (No inflmm=n)
  - pseudo papillitis
  - peripapillary telangiectasia
  - primary optic atrophy
  - papillo-macular nerve fibre bundle dropout (Nlv fibres traversing b/w OD & macula = affected)

NoR

Toxic optic neuropathy 00:50:11

- Also called as nutritional optic neuropathy, tobacco-alcohol amblyopia.
- Causes : - vitamin B1, B2, B12, folic acid deficiency.
  - methanol poisoning.
  - drugs like digoxin, isoniazid, chloroquine ☆, ethambutol. (DICE)
  - most oculotoxic ← ethambutol
- ☆☆ Visual field defect – centrocaecal scotoma > central scotoma ☆☆
- Treatment – 1000 units of hydroxycobalamin weekly for 10 weeks.

area b/w OD & macula

Colour blindness 00:56:12

- Primary colours : red : prota
  green : deutra
  blue : trita
- Types : 1. Acquired
  2. Congenital
     - Dyschromatopsia (↑ Fn)
       - Trichromatic anomaly
       - Dichromatic
     - Achromatopsia (↓ Absent)
       - Cone monochromat
       - Rod monochromat

Prota
- Anomoly : Red colour defect
- Anopia → Red colour Blindness

☆ Dueteranomoly : 3.4%
↓
Commonest among all Anomolies, Anopias

Acquired	Congenital
1. All optic nerve diseases → red-green colour blindness, except glaucoma.	1. most common mode of inheritance X-linked recessive. *
2. macular diseases → blue-yellow colour blindness, except Stargardt's disease. : cone dystrophy	2. Commonly seen in males. ☆ AIIMS
3. Old age and nuclear cataract → blue colour blindness.	3. Genes for : - Red and green → chromosome Xq28. - Blue → chromosome 7.

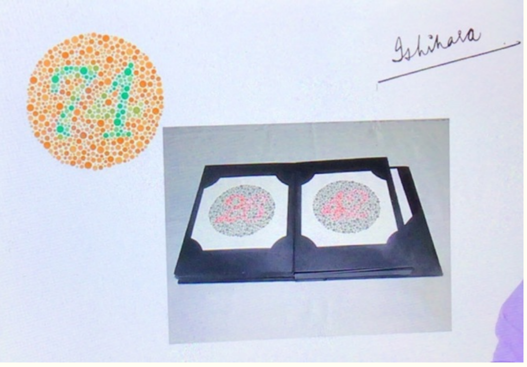
Congenital blindness			
Dyschromatopsia		Achromatopsia	
Trichromatic Anomoly	Dichromatic	Cone monochromat	Rod monochromat
All 3 colours seen, but defect in 1 colour (ex: can't diff Red.pink, orange)	2 colours seen; 1 colour anopia	Only 1 colour seen	No colour seen; : only B/W Complete blindness; Autosomal recessive

Active space

Colour blindness- Tests

1:16:53

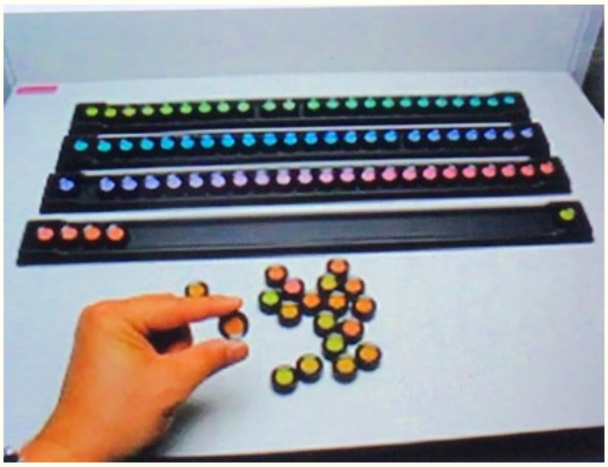
- Ishihara chart – pseudoisochromatic chart. ✓
  - most common.
  - used for screening of protan and deutran conditions. (≠ Blue)
  - consists of 38 test plates.



Id 1 colour in background of other

- Hardy Rand Riltler – pseudoisochromatic chart. ✓
  - used for screening of infants.

- Farnsworth munsell 100 hue – most sensitive test.
  - 85 test caps. (≠100)
  ☆



Active space

- Farnsworth Munsell 15 hue - 15 test caps.

- Nagel's anomaloscope -used for diagnosing severity.

- Lantern test - used for vocational testing.
- Holmgren's wool.

Papilledema

01:25:19

- Defined as the swelling of optic disc due to raised ICP (intra cranial pressure). *
- Clinical features : - always bilateral, except in Foster- Kennedy syndrome.
  - painless.
  - non inflammatory.
- Pathogenesis :

Increased ICP ⟶ Transmitted along subarachnoid space ⟶ Optic nerve (as it is Enclosed in same meningeal sheath as Brain)

↓

Leading to stasis ⟵ Blockage of axoplasmic transport

↓

mechanical obstruction ↓ Extracellular edema

Venous congestion

MECHANICAL SIGNS OF PAPILLEDEMA	VASCULAR SIGNS OF PAPILLEDEMA
Earliest sign – blurring of disc margin.	Hyperemia of O.D
Elevation of optic disc.	venous engorgement
Optic cup gets filled up.	Cotton wool spots
Edema in retinal nerve fibre layer.	Splinter H'ges on O.D
Paton's line → retinal folds around optic disc	Exudates around macula (macular star appearance)

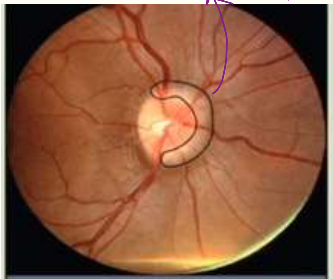
- Symptoms :
  1. Earliest : headache (worse in morning).
  2. Projectile vomiting.
  3. Vision is normal (but gradual, painless loss of vision occurs when secondary optic atrophy sets in).
  4. Amaurosis fugax – transient visual loss.
     ↳ d/t blockage of Axoplasmic Transport → No impulses to brain
  5. enlargement of Blind spot (VFD)

★ since O.D = blind spot

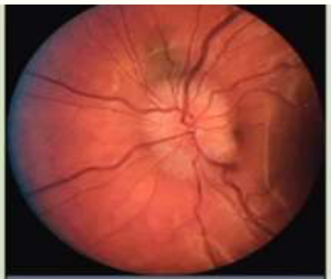
Grading of papilledema : 1:42:23

According to Frisen's scale

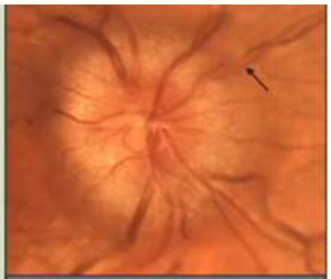
Blurring of margin



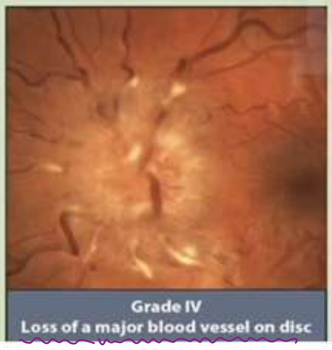
Grade I
Characterized by a C-shaped halo with a temporal gap



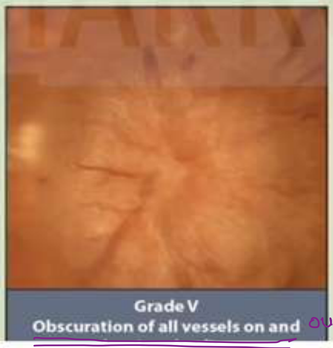
Grade II
The halo becomes circumferential



Grade III
Characterized by loss of major vessels as they leave the disc (arrow)



Grade IV
Loss of a major blood vessel on disc



Grade V
Obscuration of all vessels on and out

AION

1:45:38

O.D

- Anterior ischemic optic neuropathy
- Types :

AION

Arteritic AION | Non arteritic AION

	Arteritic AION m/c in ♀	Non arteritic AION m/c in ♂
Occurs due to	Giant cell arteritis.	Occlusion of short posterior ciliary arteries.
Laterality	Bilateral loss of vision	Unilateral loss of vision
Severity	Severe loss of vision	moderate loss of vision
Vision	<6/60	> 6/60
Premonitory symptoms (pain, amaurosis fugax)	Present	Absent
Optic disc appearance	Pale	Hyperaemic
Associations	Tender temporal artery, headache, cranial nerve palsies.	Diabetes, HTN, hyperlipidaemia. (Chr. d/s)
Treatment	Corticosteroids,✓ aspirin.✓	No treatment.

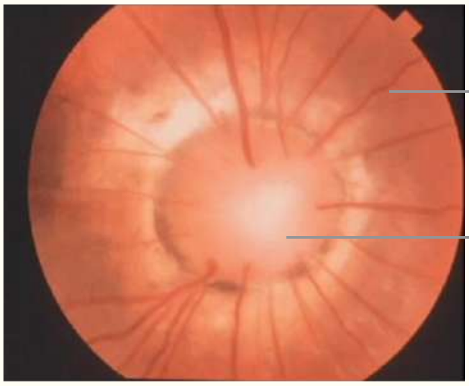
Congenital anomalies of optic disc

1:53:10

1. Morning glory syndrome

Malformation of optic disc associated with :

- peripapillary scleral defect.
- absence of lamina cribrosa.
- retro displacement of optic nerve. (No support)



Blood vessels emerge in a radial pattern

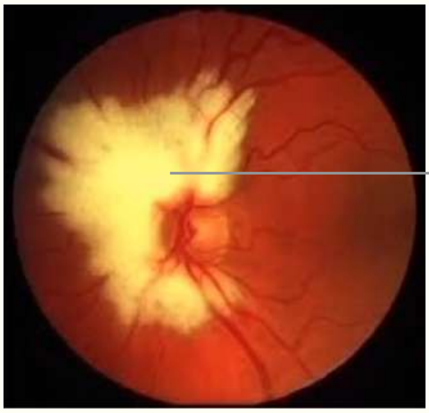
Large optic disc with excavation

2. myelinated nerve fibres

Retinal ganglion cells retain myelin sheath

↓

Visible as white streaks



Active space

SQUINT: EXTRAOCULAR MUSCLES, BINOCULAR VISION

Squint 00:00:53

Squint is when one of the eyes is deviated from its normal position.

Actions of extraocular muscles ⑦ 00:04:25

Extraocular muscle	Actions
LPS (levator palpebri superioris)	elevation of upper eyelid.
MR (medial rectus)	inward movement or adduction
LR (lateral rectus)	outward movement or abduction

Total 7 in number

- ★★ Primary actions predominate in abducted eye.
- Secondary actions predominate in adducted eye. ★★ (SAD)

in abducted eye ↑ in adducted eye ↑

	Primary action	Secondary action	Tertiary action
SR (superior rectus)	Elevation	Intorsion	Adduction
IR (inferior rectus)	Depression	Extorsion	Adduction
S.O. (superior oblique)	Intorsion	Depression	Abduction
I.O (inferior oblique)	Extorsion	Elevation	Abduction

Intorsion
Extorsion

Not clinically elicited

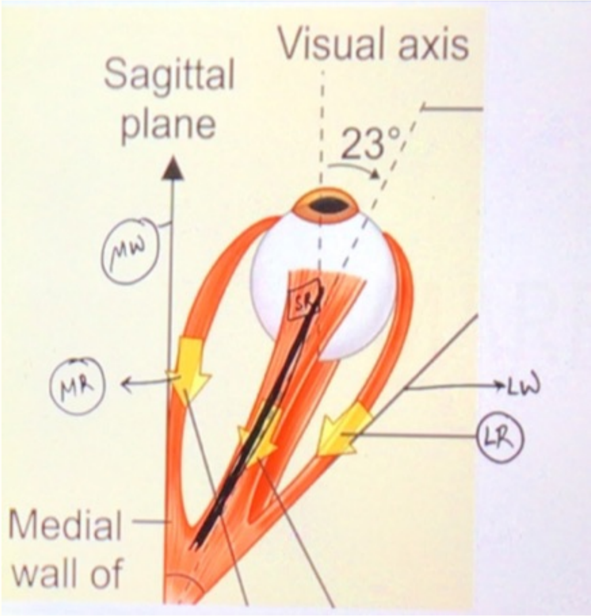
Active space

Clinical actions of extraocular muscles

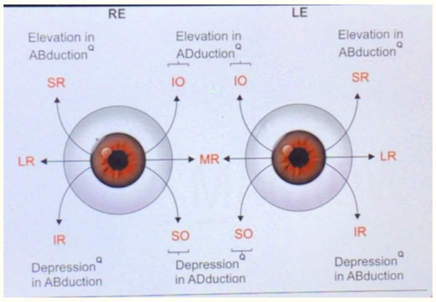
00:21:11

Extraocular muscle	Actions
SR	Elevation in Abd
IR	Depress2 in Abd
SO	Depressn in Add
IO	Elevation in Add

: Look @ tip of nose



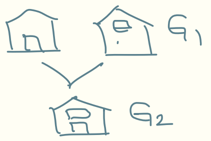
on 23° Abd
↓
V.A, m/s axis = same
↓
SR = pure elevator



Binocular single vision

00:28:31

1. Three grades :
    - G1 – simultaneous macular perception
    - G2 – fusion
    - G3 – stereopsis (depth perception)



2. The occurrence of binocular single vision includes:
    - Synergists
    - Antagonists
    - Yoke muscles/contralateral synergists
    - Hering's law
    - Sherrington's law

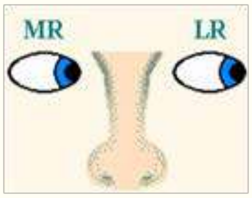
- Synergists – pair of muscles having same action in the same eye.
  example : Rt.SR and Rt.IO

Active space

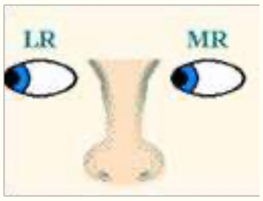
- Antagonists – pair of muscles having opposite actions in the same eye.
  example : Rt.SR and Rt.IR
- Yoke muscles – pair of muscles (one in each eye) that moves both the eyeballs together in same direction, known as version* movement or conjugate* movement.
  examples:

Yoke muscles	Action
Lt. LR and Rt. MR	Levoversion
Rt.LR and Lt.MR	Dextroversion
Rt.SR and Lt.IO	Dextro elevation
Rt.IR and Lt.SO	Dextro depression
Rt.SO and Lt.IR	Levo depression
Rt.IO and Lt.SR	Levo elevation
(Rt.SR + Rt.IO) right elevation and (Lt.SR + Lt.IO) left elevation	B/L elevation (Sursumversion)
(Rt.IR + Rt.SO) right depression and (Lt.IR + Lt.SO) left depression	B/L Depression (Deorsumversion)

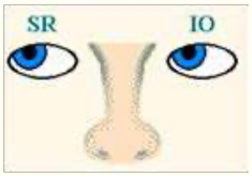
Primary gaze



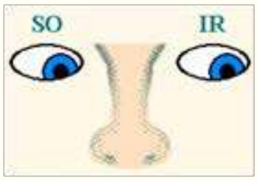
Levoversion



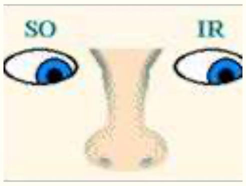
Dextroversion



Dextroelevation

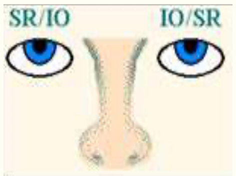


Dextrodepression



Levoelevation

Levodepression



Sursumversion

Deorsumversion

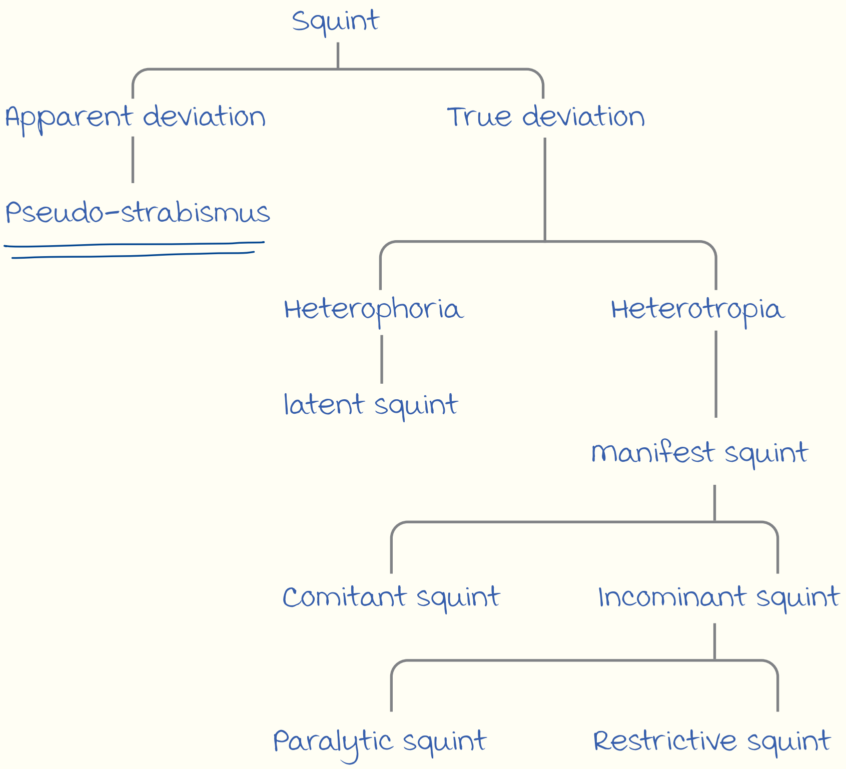
- Hering's law of equal innervation to a pair of yoke muscles – responsible for equal intensity and simultaneous neural impulse needs to reach both the yoke muscle.
  Hering's law is not followed by 'dissociated vertical deviation'.

- Sherrington's law of reciprocal inhibition – responsible for relaxation of the antagonists of the yolk muscles.

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

Squint/Strabismus

1:02:55



- Latent squint – hidden deviation which may become manifest under conditions of ocular stress.

* Example : excessive near work in dim light. *

2° deviation
↓
d/t Herring's law

Comitant squint	Incomitant squint
Degree of deviation remains same, in all directions of gaze	Degree of deviation varies with gaze
Primary deviation = secondary deviation	Primary deviation < secondary deviation
Ocular movements – normal	Ocular movements – restricted
Diplopia and head posture – absent	Diplopia and head posture – present
Sensory anomalies – present	Sensory anomalies – absent

Ⓝ eye = fixed
↓
< made by Abn eye

Abn Eye fixed
↓
< made by Ⓝ eye

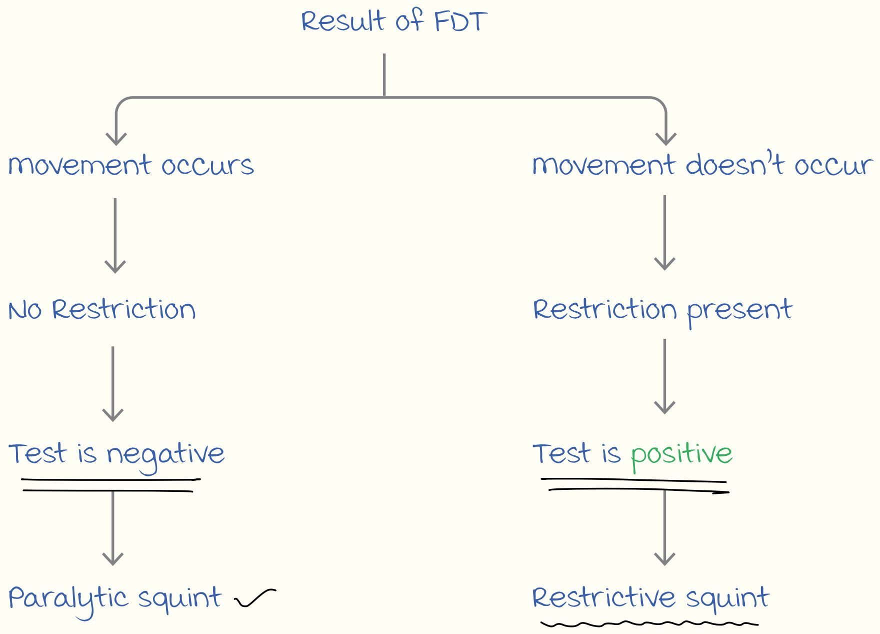
Incomitant squint :

1:20:45

FDT (forced duction test) is done to differentiate between paralytic and restrictive squint.

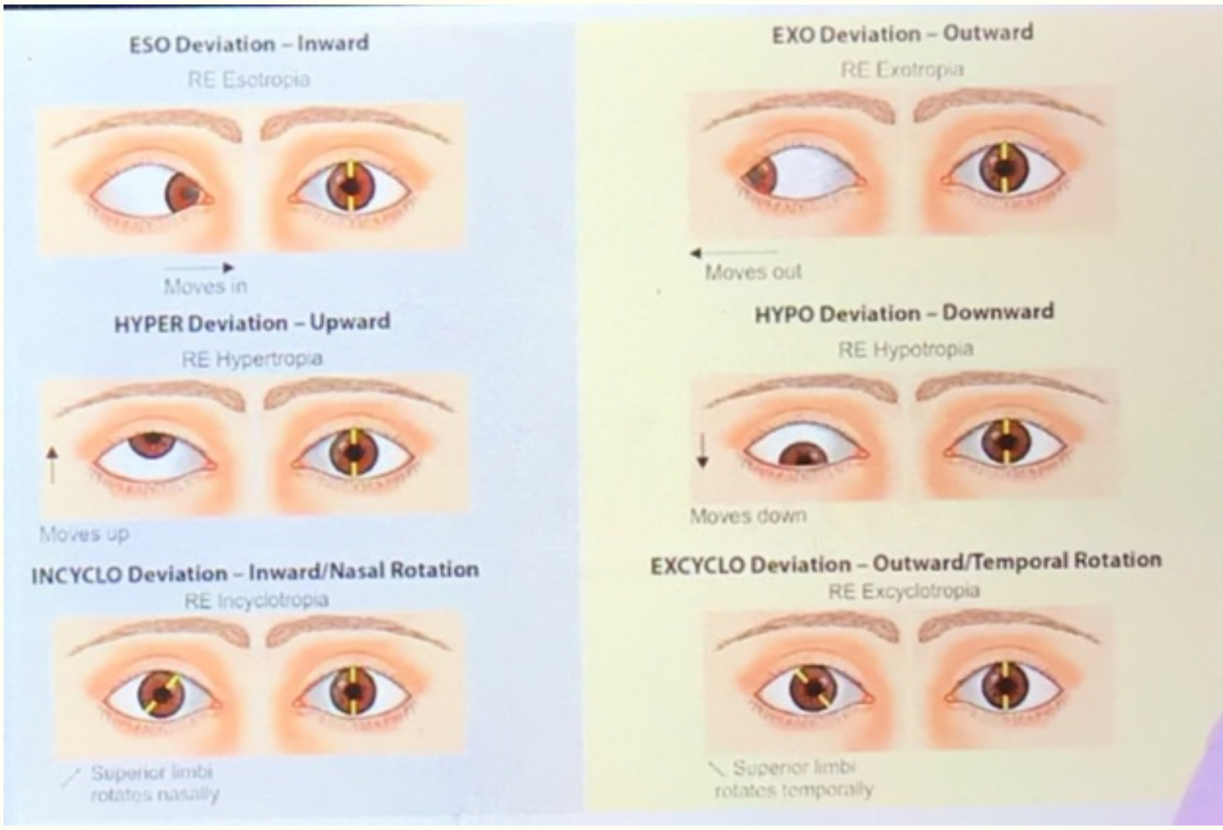
FDT is diagnostic for restrictive quint. (Test = diagnostic only if ⊕)

method : hold the eye with forceps and then move it with external force.



Directions of deviations 1:26:34

- Esodeviation
- Exodeviation
- Hyperdeviation
- Hypodeviation
- Incyclodeviation
- Excyclodeviation



ESO Deviation – Inward
RE Esotropia
Moves in

HYPER Deviation – Upward
RE Hypertropia
Moves up

INCYCLO Deviation – Inward/Nasal Rotation
RE Incyclotropia
Superior limbi rotates nasally

EXO Deviation – Outward
RE Exotropia
Moves out

HYPO Deviation – Downward
RE Hypotropia
Moves down

EXCYCLO Deviation – Outward/Temporal Rotation
RE Excyclotropia
Superior limbi rotates temporally

Active space

SQUINT: TESTS

Examination of Squint 00:00:19

① Examination of ocular movements in all the directions of gaze.

② Visual acuity assessment and Refraction.

Visual acuity assessment ↓ Snellen's Chart

Refraction ↓ refractory power/ Spectacle power to be prescribed

↓

Retinoscopy/Skiascopy : For Retinoscopy, use Mydriatic-Cycloplegics * → Paralyse Ciliary m/s ↓ Abolish Accomodation

- Drug of choice for children<5 years :

Strong Cycloplegic ← 1% Atropine Ointment ↳ ↑↑↑ Accomodation

- Drug of choice in adults :

0.5% or 1% Cyclopentolate Drops

Accommodative Squint 00:06:30

Uncorrected hypermetropia leads to Esotropia

↓

(Loss of near Vision)

↓

Leads to over accommodation and over convergence

↓

Presents as Esotropia

Treatment : Spectacles

③ Measurement of Vergence : Extrapower by each m/s to keep the eye in straight position

Vergence → Different movement of eyeballs < Convergence / Divergence

A. Synoptophore

B. Livingstone gauge

C. Pencil Test

D. RAF ruler- Royal Air Force

- Target is moved forward or towards the patient
- We note the distance at which the patient reports Diplopia
- This distance is called as NPC - Near Point Of Convergence

⇒ • NPC < 10cms : Ⓝ

RAF ruler

RAF ruler

4. Measurement of Ocular Deviation

Objective Test	Subjective Test	
• Hirschberg,s test • Bruckner,s test • Krimsky test • Cover – Uncover test • Prism bar cover test	• (Based on Diplopia principle) • Maddox rod • Maddox wing • Tangent screen	• (Based on Haploscopic princeple) • Lancaster R –G test • Synoptophore

Objective test

00:17:32

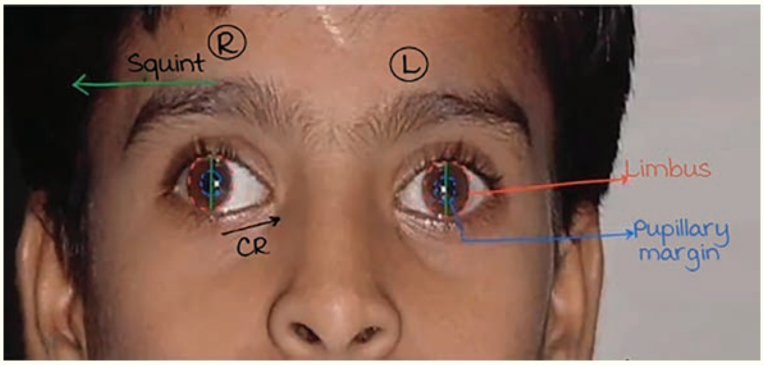
A. Hirschberg's (Corneal Reflection) Test

↓

2 Uses

Used to find out Direction of deviation

Used to find out Degree of deviation



Cr – inward Nasal to center of pupil

CR – center of pupil

Active space

* Direction of Deviation

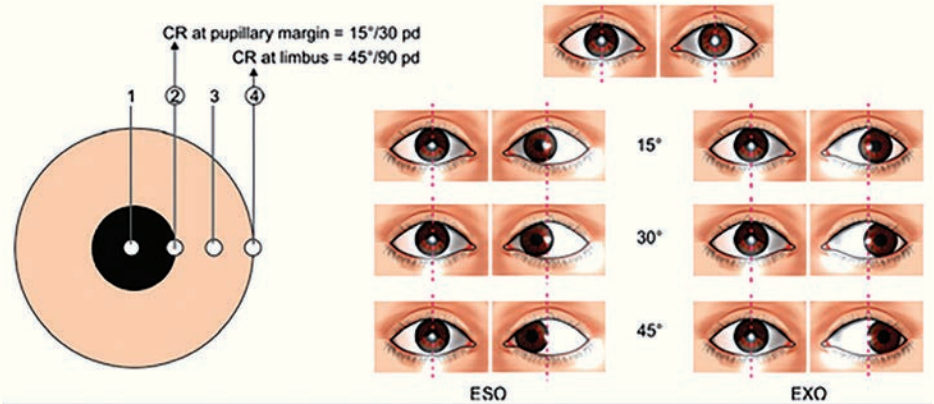
↓

opp to direction of Reflection

* Degree of Deviation :

1 mm of deviation of CR from center of pupil = 7° squint | 14 prismdiapter squint (pd)

$1 \text{ PD} = \frac{1}{2}^{\circ}$



Clinical rule (HCRT)

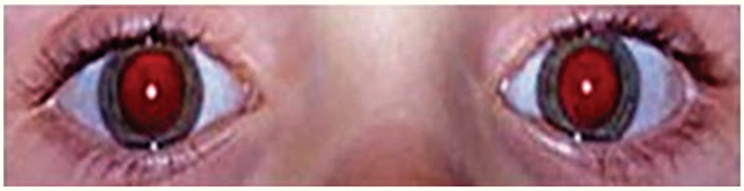
Q) case scenario: Patient presents with a squint, on examination corneal reflection is seen 2mm nasal to the center of the pupil. Diagnose.

A) 14-degree Exotropia

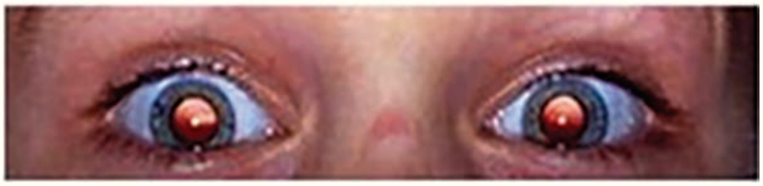
Bruckner's test

00:33:09

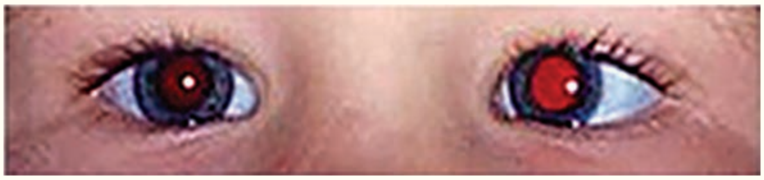
- Used to screen squint and amblyopia in "Infants"
- Done using ophthalmoscope by accessing the red fundal reflex.



Case 1 : Color and brightness are equal in both eyes. Normal.



Case 2 : Crescents of different brightness in each eye. Refractory error. → Can lead to Amblyopia



Case 3 : Eye with a brighter color, has Squint. The left eye has a squint in the given image.

Cover-uncover test

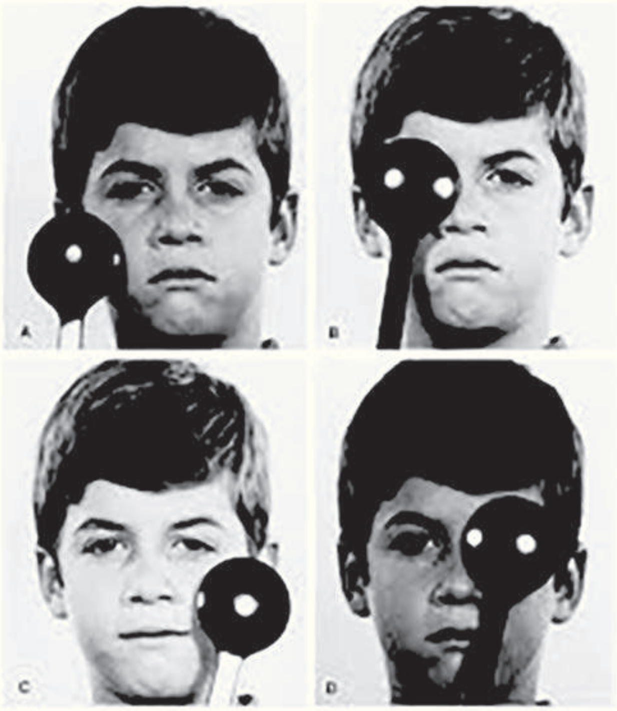
00:37:06

Cover Test
↓
Used for confirmation of Tropia

"CT"

Uncover Test
↓
Used for diagnosis of Phoria

"UP"



Cover -uncover test

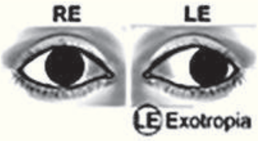
D - Deviation
O - Opposite
O - Of
M - Movement

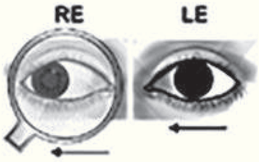
Case scenario : On the Right eye cover test, the Left eye moves inwards

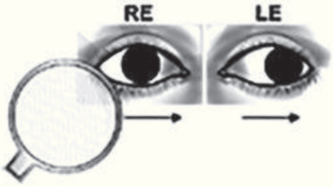
Diagnosis : Left eye Exotropia

Case scenario : On the Right eye un-cover test, the right eye moves upwards.

Diagnosis : Right eye hypo Phoria







Active space

Case 1 : Cover test done

↓

→ Covered the normal eye (right eye in this case)

↓

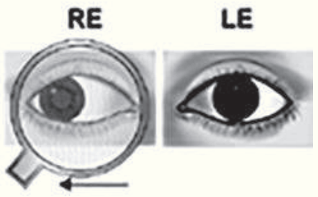
⇒ The Deviated eye (left eye in this case) moves inwards to pick up the fixation

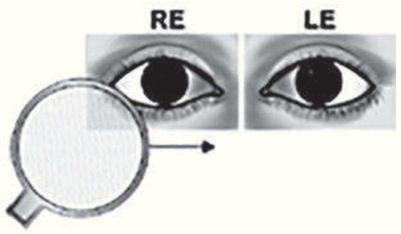
↓

Thus, EXO-eye moves inwards on the cover test.

↓

Again Normal position is regained





Case 2 : Patient can be Normal or have "Phoria".

↓

The Uncover test is done.

↓

The right eye is covered, but the left eye does not move as already fixated on an object.

↓

On Uncover of the right eye, the right eye moves inwards

↓

Denoting that right eye had moved outwards behind the cover.

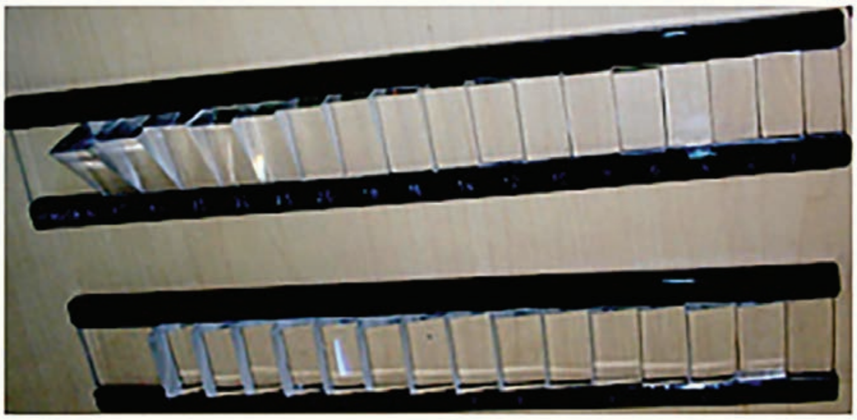
↓

Thus Exophoria.

Active space

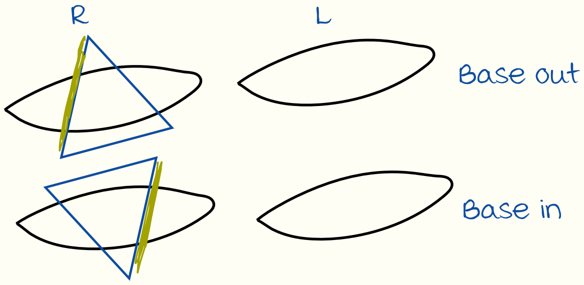
Prism bar cover test [PBCT]

00:53:35



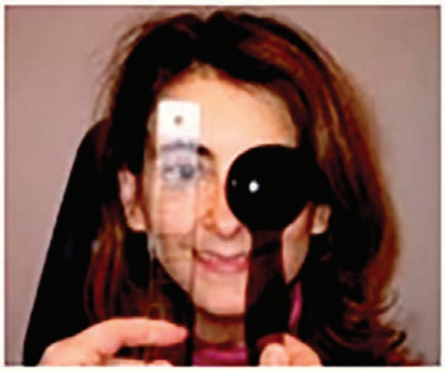
"The best test for measurement of deviation (degree) in Tropia"

method : Prisms are placed in front of the deviated eye.



D – Deviation
O – Opposite
O – Of
B – Base of the prism

- * ESO – Base Out Prism ✓
- * EXO – Base In Prism
- * Hyper – Base Down Prism



Perform the alternate cover test till the deviated eye does not show any movement- "End Point"

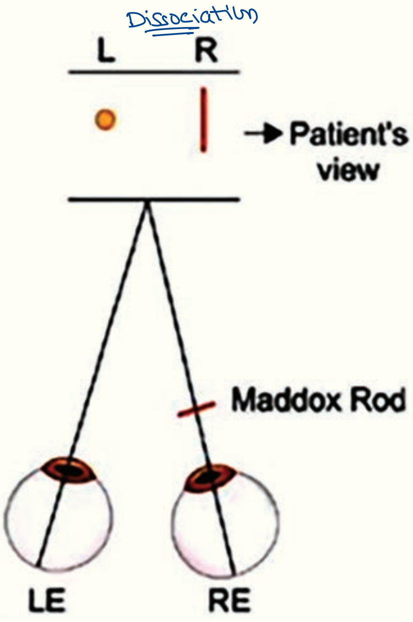
Subjective Test

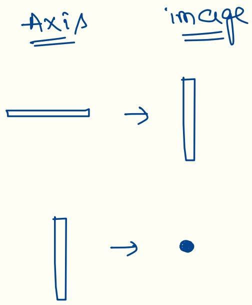
Maddox rod test

00:59:55

Red, Plano-convex (striated) lens

1) To measure degree of phoria @ far point of Fixation
2) Macular fn test
3) Double maddox rod test ⇒ Dx of Cyclodeviation (SO palsy)

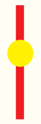




Active space

Results

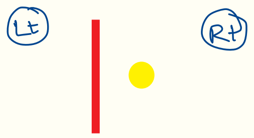
a) Normal Eye



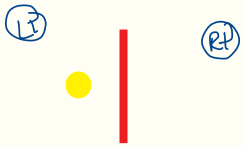
: fusion ⊕

X

b) Crossed diplopia : Exophoria

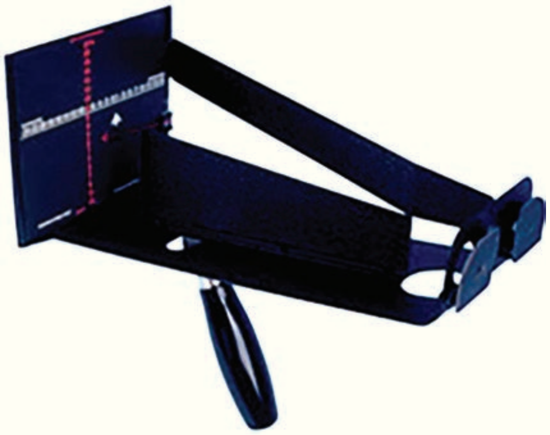


c) Uncrossed diplopia : Esophoria



Maddox Wing

01:07:50



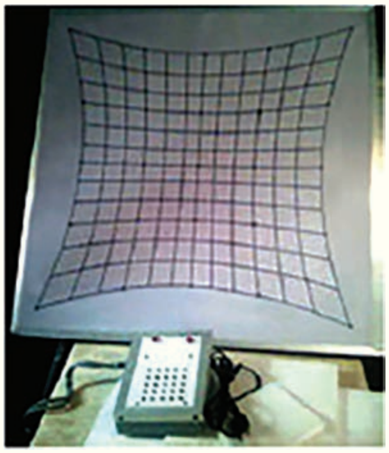
It is used to measure amount of Phoria at near point.

Tests for diplopia

01:08:55

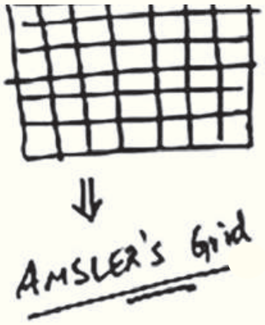
I. Red-Green Goggles
II. Hess Chart
III. Lee's Screen

Hess Chart : does not have straight lines

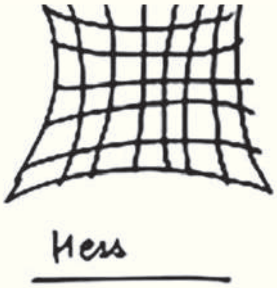


All lines = concave

Amsler's Grid vs. Hess Chart



Amsler's grid

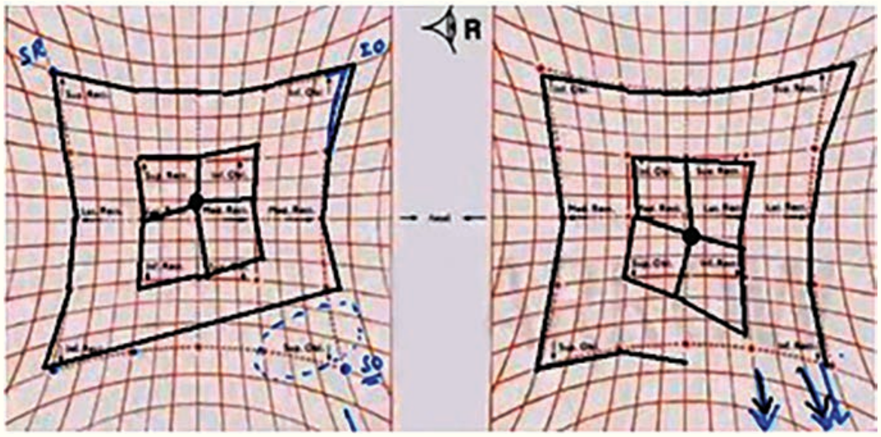


Hess

Things to see in a Hess Chart –

- Deficiency of action : "SO" action : ↓
- Over action of the antagonistic in the same eye : "IO" : action ↑
- Over action of the yolk muscle in the other eye : "IR' c/L : ↑↑ Action

Case :



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In this case the patient has not been able to perform depression in adduction because the superior oblique action should have been up till the blue dot "SO". Therefore the antagonist muscle i.e. inferior oblique in the same eye is overacting (slightly to the right. Also the yolk muscle of other eye starts to overact i.e. inferior rectus in this case.

Diagnosis : Diplopia in Downgaze (superior oblique of left eye)

Tests for sensory anomalies

01:13:40

This group of test differentiates between :

a) Binocular single vision
- I object, I image.

b) Anomalous retinal correspondence(ARC) ↓ : Fovea of one eye Corresponds to Extrafoveal point of other eye : as adaptation
- Occurs in cases of Diplopia(as an adaptation)
- I object, 2 images
- Diplopia occurs in small angle squint

↓

Image in both eyes formed at non corresponding points

c) Suppression
- Occurs in cases of Confusion(as an adaptation)
- 2 objects, 2 images
- Occurs in large angle squint

↓

✱ Suppression of deviated eye ✱

↓

✱ Amblyopia(loss of vision without any organic cause)

Test :

iv. Worth 4 dot test

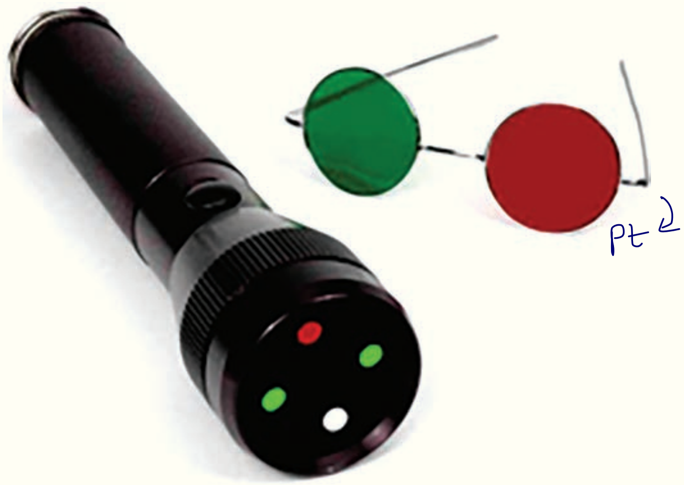
v. striated glasses

vi. Synoptophore

vii. 4 ▲ (prism dioptre) cover test- Diagnosis of microtropia(<5°) ☆

Worth 4 Dot Test

01:24:20



Pt ↲

Color of dots :

- 1 Red
- 2 Green
- 1 White

method :

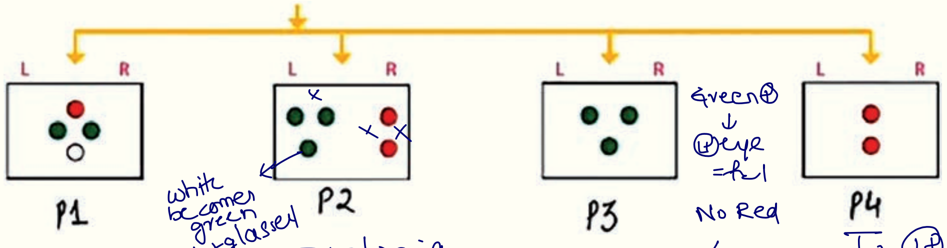
Patient wears "Red-Green" Goggles. Red on right eye and green on left eye.

Rt ↑ Lt ↑

↓

Patient asked to look at 4 dots

Results :



white becomes green cause glasses

- Binocular single vision
- ARC (harmonious)

Diplopia or Non Harmonious ARC

Green ⊕ ↓ Lt eye = fn 1

No Red ↙

Rt eye = suppress

Red ⊕ ↳ Rt eye = fn 1

No Green ↓ ↳ Lt eye = suppressed

Active space

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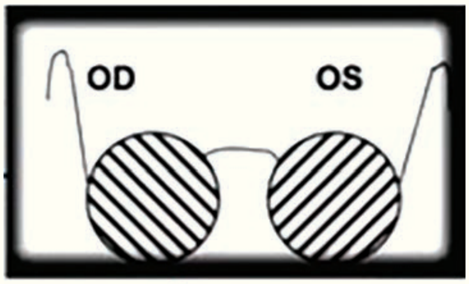
2) Bagolini Striated glasses



45° 135°

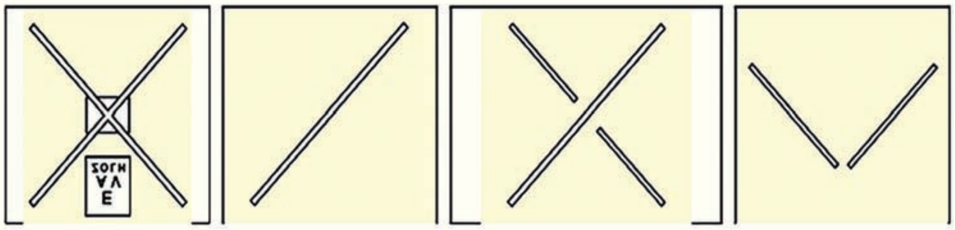
Have oblique striations.

method: Patient wears glasses

↓

450 Axis – line visible as 135°

1350 Axis – line visible as 45°



No fusion

- Binocular single vision
- Harmonious ARC

· Suppression

Suppressive central scotoma

· Diplopia

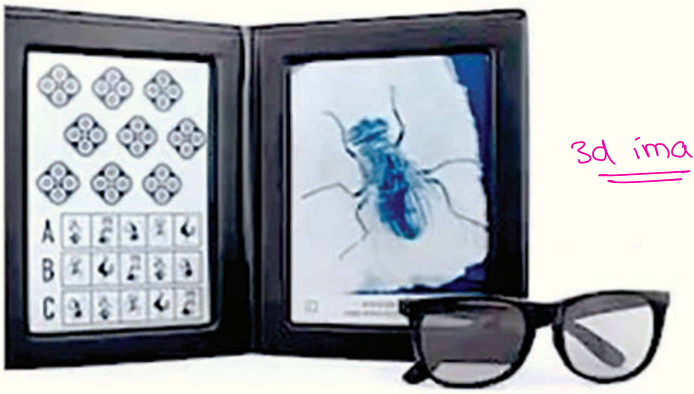
· Non Harmonious ARC

→ Grade 3 BSV : depth

Tests for stereopsis

01:36:00

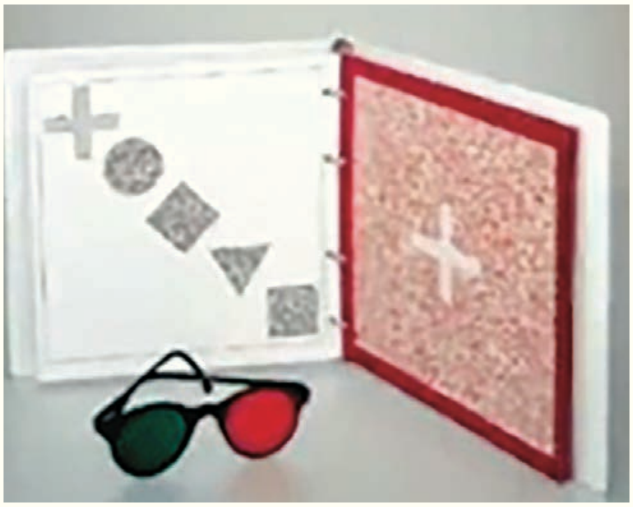
i. Two pencil test
ii. Synoptophore
iii. Titmus Fly Stereo Test[TFST] :



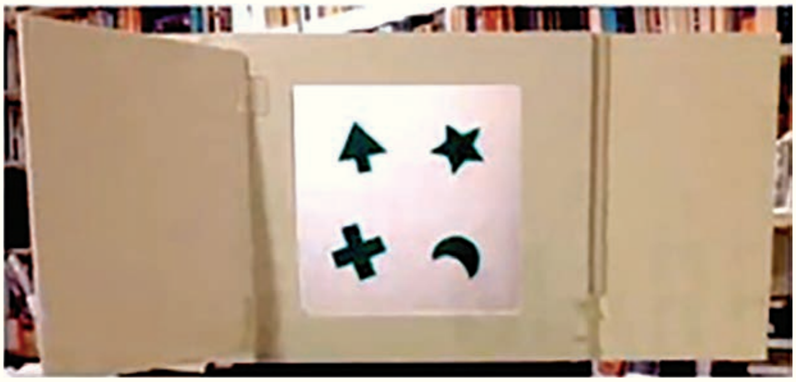
3d image

IV. Random Dot Stereo Test: Better than TFST because these tests eliminate the use of mono-Ocular cues to depth.

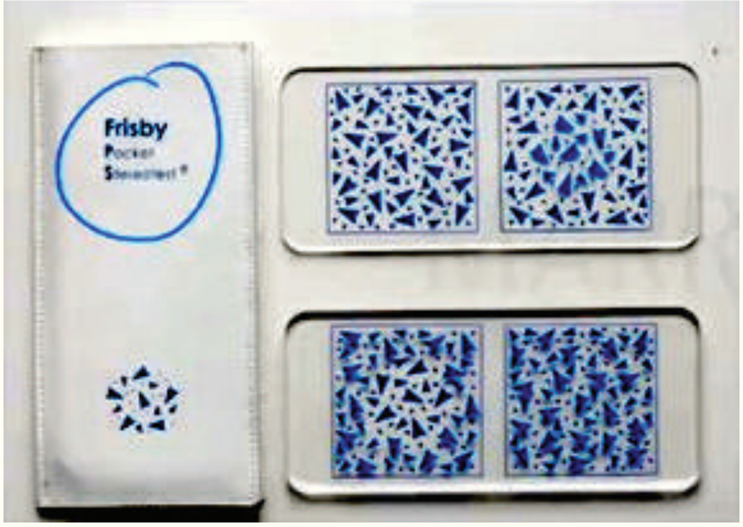
a) TNO test



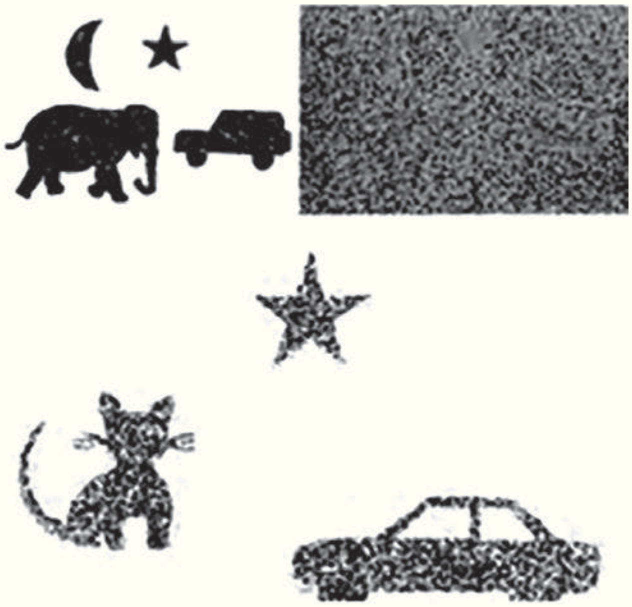
b) Frisby Davis Test



c) Frisby test



d) Lang Test



Active space

SQUINT: PARALYTIC SQUINT

Paralytic Squint

00:00:57

Paralytic squint occurs when there is an acquired defect of movement of an eye. Occurs due to disease of the III, IV and VI cranial nerves.

IIIrd cranial nerve or, oculomotor nerve palsy

- Course :

Nucleus (in mid-brain at level of superior colliculus)

↓

IIIrd CN enters lateral wall of cavernous sinus

↓

Passes through superior orbital fissure with in annulus of Zinn

- Nucleus has 2 parts :

Main motor nucleus

↓

Supplies extraocular muscles (5)

↓ Superior rectus, inferior rectus, internal oblique, medial rectus

↓

Supplied contralaterally

↓ Levator palpebre superioris

↓

Supplied by an unpaired nuclei

(single)

Edinger-Westphal nucleus

↓

Supplies sphincter pupillae * (2)
muscle & ciliary muscle *

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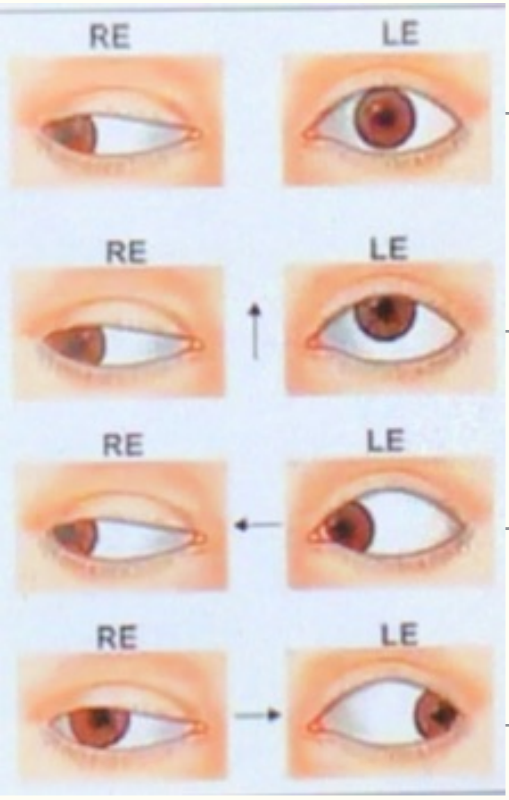
- Clinical features of IIIrd cranial nerve palsy :
  1. Ptosis. [x LPS]
  2. Hypotropia. (x SR, IO)
  3. Exotropia. (x MR)
  4. mydriasis (no light reflex). [x S·P]
  5. Loss of accommodation (no near reflex). [x ciliary m/s]
  6. Crossed diplopia.



→ Ptosis + down & out eye

→ Limitation of elevation

→ Abduction +ve

→ Adduction –ve

Special clinical features :

00:15:49

i) DM/HTN : 3rd CN palsy $\bar{c}$ pupillary sparing (LR ⊕ NR ⊖)

vasc. d/s
↓
Affect Central v/s
↓
spare peripheral N/s fibres (supplying Sp. pupillae)

ii) Uncal herniation : only Mydriasis
↓
Compressive : peripheral N/v fibre inv. : s. pupillae involved

iii) Benedikt's Sx : 3rd CN palsy (+) Flapping tremors (C/L extrapyr. signs)
[BFR]
↓
lesion through Red Nucleus

iv) Weber's Sx : 3rd CN palsy + C/L Hemiparesis
(WCH)
↓
lesion through cerebral peduncle

Telegram--@latestpgnotes

v) Nuclear 3rd CN palsy:

Daroff's rule ← B/L ptosis (unpaired Nuclei for LPS)

C/L SR weakness

Treatment for III CN palsy

00:24:32

1. Observation for at least 6 months. *

(as IIIrd CN can regenerate) :

↳ covered by Neurilemma { Schwann cells (PNS)

oligodendrocytes (CNS)

2. Surgery.

Aberrant regeneration

Pseudo Von- Graffe sign

↓

Occurs due to fibres of inferior rectus misdirected to LPS

IR→LPS

↓

On downgaze, both inferior rectus and LPS contracts

↓

Thus, looks like lid- lag

*

Psedo ARP (accomodation reflex present)

↓

Occurs due to fibres of ciliary muscle misdirected to sphincter pupillae + fibres of sphincter pupillae completely damaged

Ciliary m/s → S. pupillae

× S. pupillae

↓

Accommodation reflex present and, pupillary light reflex is absent

Near reflex ⊕

Light reflex ⊖

Von- Graffe

↓

Lid lag

(lid lags behind ball)

ARP

Argyll Robertson pupil

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

00:33:47

Trochlear N/v :-

- Course :

Nucleus (in mid-brain at level of inferior colliculus)

↓

Leaves mid-brain on the dorsal surface after decussation (internal)

↓

Enters cavernous sinus on lateral wall but,
passes through superior orbital fissure outside annulus of Zinn

↓

Supplies contralateral superior oblique muscle

- Clinical features of IVth cranial nerve palsy:

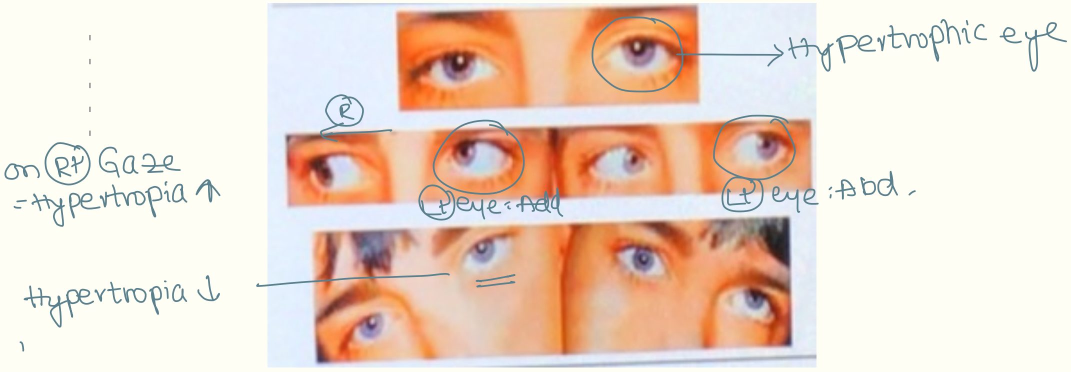
1. Excyclotorsion
2. Hypertropia.
3. Increased hypertropia on adduction/ increased hypertropia on opposite gaze. (Lt eye → toward Rt side)
4. Vertical diplopia. (C/o diplopia while climbing downstairs)
5. Head posture – Head performs same action as paralysed m/s
    a. For depression – chin depression.
    b. For intorsion – head tilt towards opposite side.
    c. For depression in adduction – face turn towards opposite side., chin depressn

Treatment for IV CN palsy :

00:49:30

Surgical (according to Knapp's classification).
most common procedure is inferior oblique weakening.

- For diagnosis of any superior or inferior muscle palsy :



Active space

Park's 3 step test

Step 1 :
Identify the hypertropic eye

Step 2 :
Hypertropia increases in direction of clinical action of paralysed muscle, either in right gaze or left gaze

⇒ for SO - Hypertropia -↑ on opp. gaze

Step 3 :
Hypertropia decreases on tilting of head in same direction of cyclical action of paralysed muscle

SO → Intorsion (opp side head tilt)

BOOT : Better on opp Tilt

VI Cranial Nerve palsy

WOOG : Worse on opp-gaze

00:59:47

- Course :

Nucleus (situated in pons)

↓

On exit from brain stem, it passes through Dorello canal

↓

Enters cavernous sinus, runs in lateral wall then passes through superior orbital fissure inside annulus of Zinn → 3,6

↓

Supplies ipsilateral lateral rectus muscle

- Clinical features of VIth cranial nerve palsy :

1. Esotropia.
2. Uncrossed diplopia.
3. Head posture – face turn towards same side.
4. ⇒ Ipsilateral horizontal gaze palsy.

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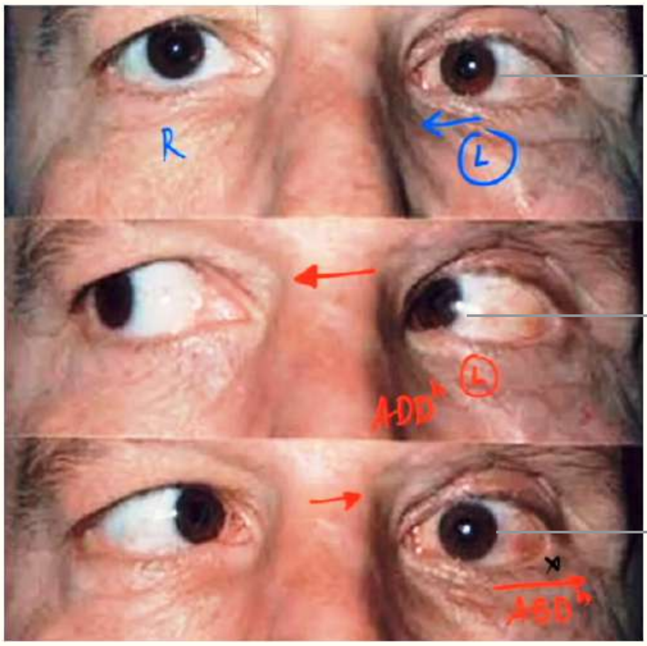


Left eye esotropia

Left eye adduction is normal

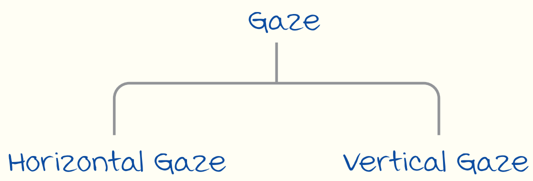
Left eye adduction is absent

- Causes of VIth cranial nerve palsy :
  ↳ 4 Nlvs : 5, 6, 7, 8
  1. Foville syndrome - ipsilateral V+VI+VII+VIII cranial nerve pal with central Horner's syndrome. (1st order Neurons)
  2. Millard Gubler syndrome – ipsilateral VI+VII cranial nerve palsy with contralateral hemiplegia.
  3. Moebius syndrome – bilateral VI+VII cranial nerve palsy.

- All clFs : opp. to m/s paralysed
- Head posture : same to action of m/s paralysed
- Squint ↑ in the direction of action of m/s paralysed

Active space

SQUINT: GAZE PALSY, RESTRICTIVE SQUINT, COMITANT SQUINT & MISC.



Definition

00:00:25

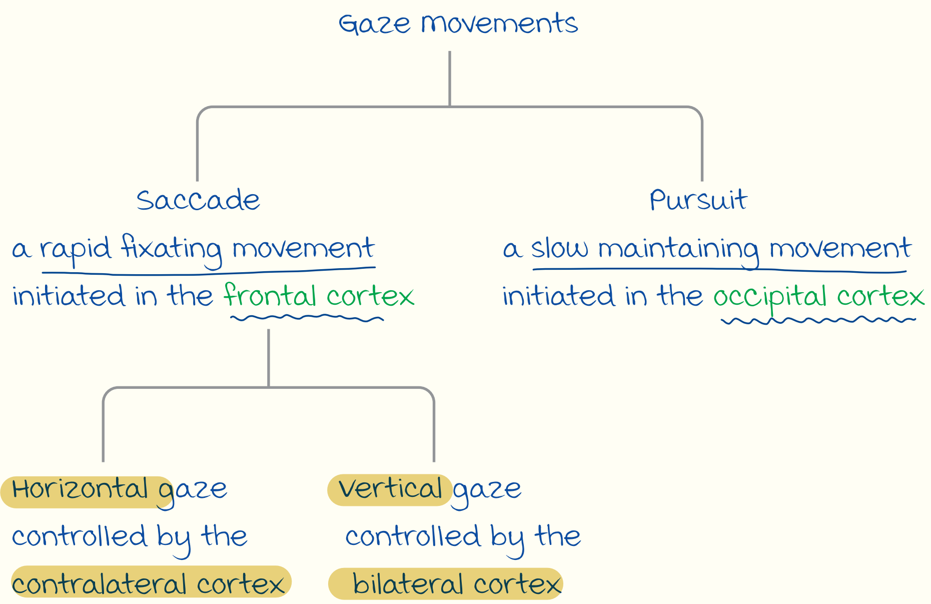
Controlled by two muscles (Supranuclear gaze control centers in the cortex give inputs to the nucleus of the cranial nerve 3, 4,and 6) leading to binocular vision.

Ipsilateral lateral rectus (controlled by the sixth cranial nerve)

Contralateral medial rectus (controlled by the third cranial nerve)

Gaze Movements

00:01:05



Saccade: a rapid fixating movement initiated in the frontal cortex

Pursuit: a slow maintaining movement initiated in the occipital cortex

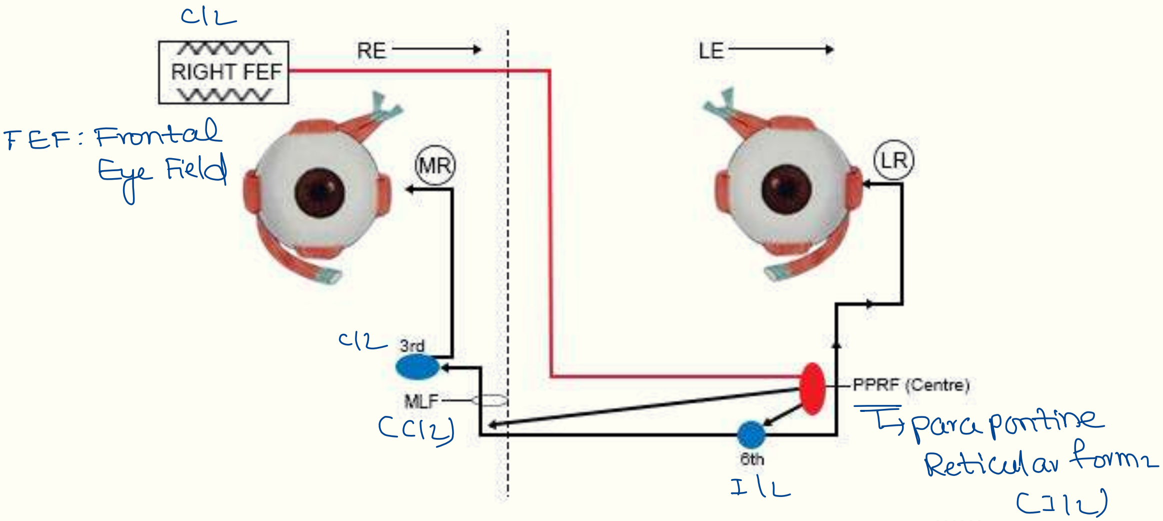
Horizontal gaze controlled by the contralateral cortex

Vertical gaze controlled by the bilateral cortex

Active space

Horizontal gaze pathway 00:03:37

This can be understood by taking the example of a single gaze i.e. left gaze.



Active space

The structure involved in the horizontal gaze :

Contralateral FEF

Ipsilateral PPRF

Contralateral MLF

Note : MLF has no role in the lateral rectus and it only controls the medial rectus.

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Horizontal gaze lesions

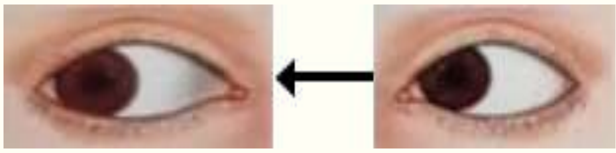
00:07:55

There can be lesions in the left horizontal gaze pathway at these three sites.

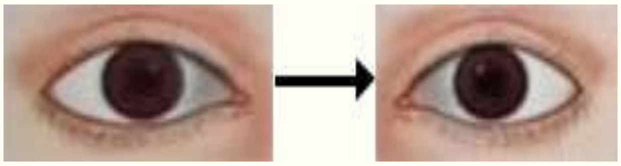
- [Contralateral FEF] Right FEF lesion : leads to left gaze palsy.
- [Ipsilateral PPRF] Left PPRF lesion : leads to left gaze palsy.
- [Contralateral MLF] Right MLF lesion : causes left gaze DEFECT.*

Case number 1 :

Primary Gaze, with both eyes looking forward. Normal.



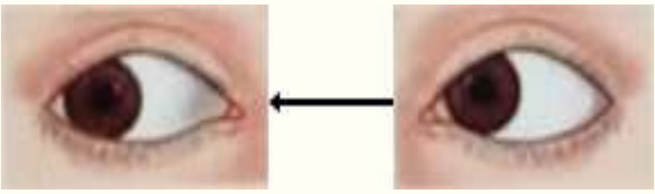
Right Gaze, as both the eyeballs have moved towards the right side so no defect.



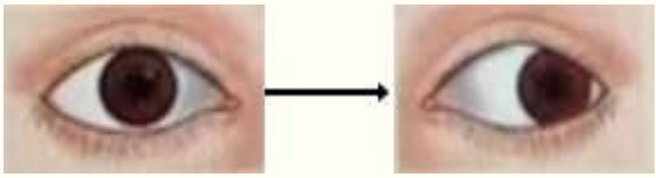
Left Gaze, but none of the eyeballs are moving, therefore, left gaze palsy. It can occur both due to the right FEF lesion and left PPRF lesion.

Case number 2 :

Primary Gaze, with both eyes looking forward. Normal.



Right Gaze, as both the eyeballs have moved towards the right side so no defect.

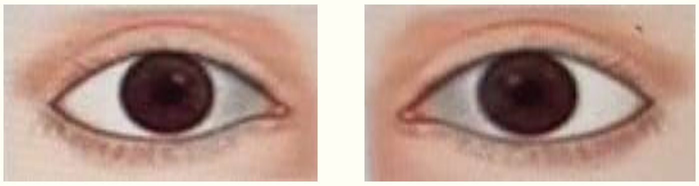


Left Gaze, left eye abduction is present but the right eye has not been able to adduct, so there has to be some problem with the right medial rectus. Since only one muscle is getting affected and that is the medial rectus so the lesion is in the right MLF.*

Left gaze defect.

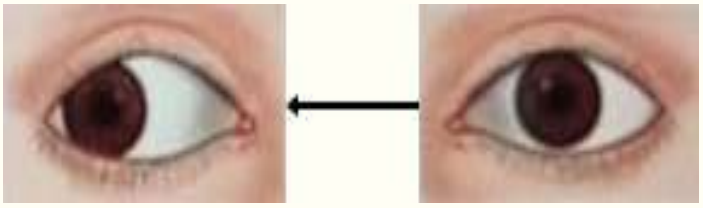
⇒ Note: The left eye abduction in this condition is accompanied by nystagmus movements.

Case number 3 :



Primary Gaze, with both eyes looking forward. Normal.

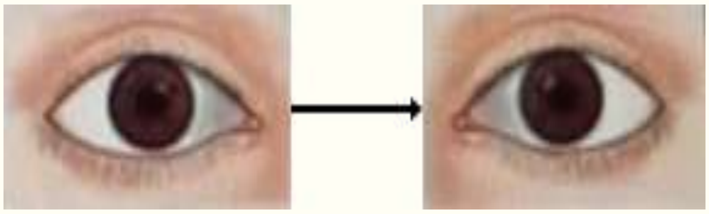
Primary Gaze, with both eyes looking forward. Normal.



1/2

Active space

Right Gaze, the right eye has been able to move but the left eye has not been able to move. Left eye adduction is absent so it Left eye MLF lesion. But wait, before moving to the conclusion see the last image for a complete diagnosis.



①

Left Gaze, none of the two eyes have been able to move towards the left side. Complete left gaze palsy. Left PPRF lesion.

Note : On the left side, two structures are damaged the left MLF and the left PPRF. The PPRF lesion is leading to complete palsy of the left side and the left MLF lesion is leading to a partial defect on the right side. In this case, out of two sides, one and a half sides are lost (one complete left side and half right side). This is known as One and a half syndrome

Vertical gaze pathway

00:17:05

Vertical gaze

Primary Centre

rimLF :Rostral interstitial medial Longitudinal Fasciculus.

Secondary Centre

INC :Interstitial nuclus of Cajal

★ AIIMS

Vertical gaze Palsy

00:18:33

a. MCC- Distal basilar artery ischemia

b. Progressive supranuclear Palsy (PSP)

- Old age ✓
- Initially - Downgaze Palsy

↓

Upgaze Palsy

↓

Global gaze Palsy

↓

Convergence Palsy

c. Parinauds Dorsal Midbrain Syndrome

- Cause : Pinealoma
- Site of lesion : Posterior Commissure (connects both rimLF)
- c/f :
  - Supranuclear "UPGAZE" Palsy ✓
  - Light near dissociation < ARP, PDMS
    (Light reflex~absent; Near reflex~present)
  - Collier's sign : upper eyelid retraction

Restrictive Squint

00:23:43

Due to external restriction, not due to some palsy.

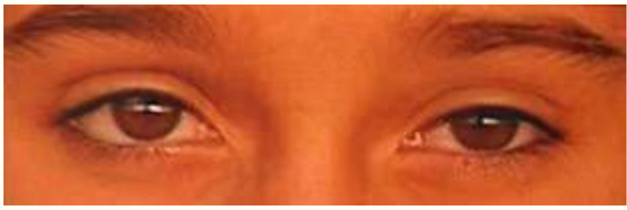
Diagnosed by FDT (Forced Duction Test)

DRS(Duane's Retraction Syndrome)

00:24:34

c /f → Types ↓	a) Limitation of movement	b) Retraction of Globe ↓ c) Closure of palpebral fisure
1	Abduction B	on Adduction D
2	Abduction B	on trying to perform Adduction D
3	Abduction & Adduction B&D	on Abduction & Adduction B & D

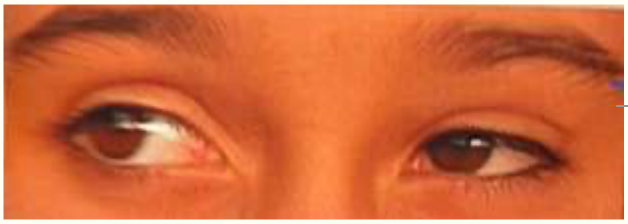
CASE :



→ 1° Gaze

=Ⓝ : i.e cannot by 6th N/v palsy

Normal



→ Ⓡ Gaze

Palpebral fissure closure on Add^n (D)

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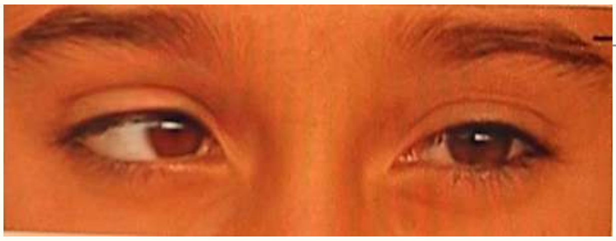
The right eye and left eye able to move towards the right side but in the left eye, closure of palpebral fissure on adduction.



→ (L) Gaze

limitation of Abdz (B)

Left eye abduction is limited

So this is a case of Type I DRS.

Brown's Syndrome 00:34:05

Due to mechanical restriction caused by a congenitally tight / fibrosed Superior Oblique (SO)

↓

SO os contracted → CANNOT Relax

↓ [Sherrington's law]

Inferior Obliques (IO) is unable to CONTRACT

Presents with Absence of Elevation IN ADDUCTION (Clinical action of IO)

Comitant Squint 00:36:55

- usually presents as Esotropia

Comitant Esotropia

- Accomodative
  ↓
  - Refractive
    - Hypermetropia → Esotropia
    So Hyper
  - Non Refractive
    - ↑ Convergence
      or
      ↓ accomodation
    - Esotropia : near > far
- Non Accomodative

Active space

- T/t :

a. Spectacles(Bifocal)

b. miotics like Ecothiophate *

c. Surgery like :

- Bimedial recession
- FADEN Sx(retro equatorialmyopexy)

Non-Accommodative Esotropia

No cause ←

"Essential INFANTILE Esotropia" #PG1

Associated with :

⇒
- Latent Horizontal Nystagmus
- IO overaction
- Dissociated vertical deviation

⇒ Always a LARGE ANGLE SQUINT i.e. → Suppressn → Amblyopia (>30 D)

Treatment :

1) Prevent Amblyopia

a) Occlusion therapy: Patch normal eye *

method : cyclical patten.

⇒ • Occlude the normal eye for days = Age of the child in year ↓f/b 5y : 5d

⇒ • Occlude the deviated eye = 1day

b) Penalization : Use atropine drops in the normal eye ⇒ dilated

↓

doesnot constrict

↓ on sun light

Photophobia

↓

Closes eye

2) Surgery

Recession and → Lengthening → WEAKENING

Resection → Shortening → STRENGTHENING

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LENS: ANATOMY AND METABOLISM

Introduction to Lens

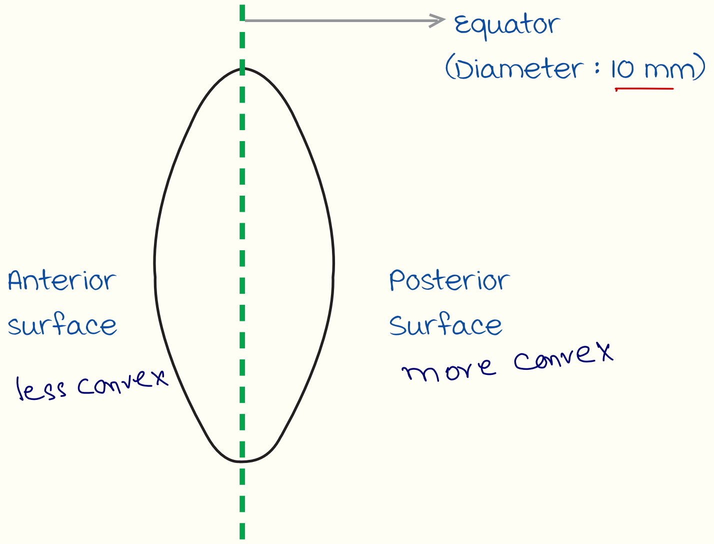
00:01:43

Colour : Transparent

Blood supply : Avascular

Nerve supply : No innervation

Shape : Biconvex



- Anterior surface : Less convex, flatter.
  Radius of curvature : 10 mm.
- Posterior surface : more convex, curved.
  Radius of curvature : 6 mm.
- Equator diameter : 10 mm.
- Anteroposterior diameter :

Position / site of lens : Patellar fossa

Refractive power of lens : +16D to +19D.

Refractive index of lens :

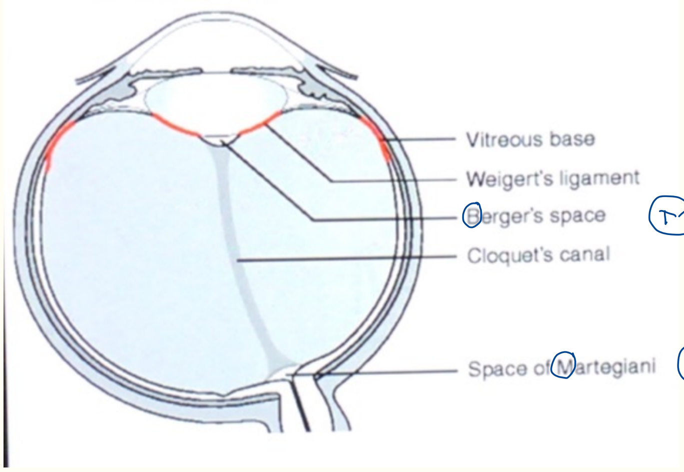
- Central part : 1.406 (Part of the eye with maximum refractive index).
- Peripheral part : 1.386

Active space

Lens develops from : Surface ectoderm (only inner structure)

Lens and associated structures

00:11:50



Mittendorf dots

Bergmeister's papillae

Weigert's ligament : Attachment between posterior surface of lens and anterior face of vitreous.

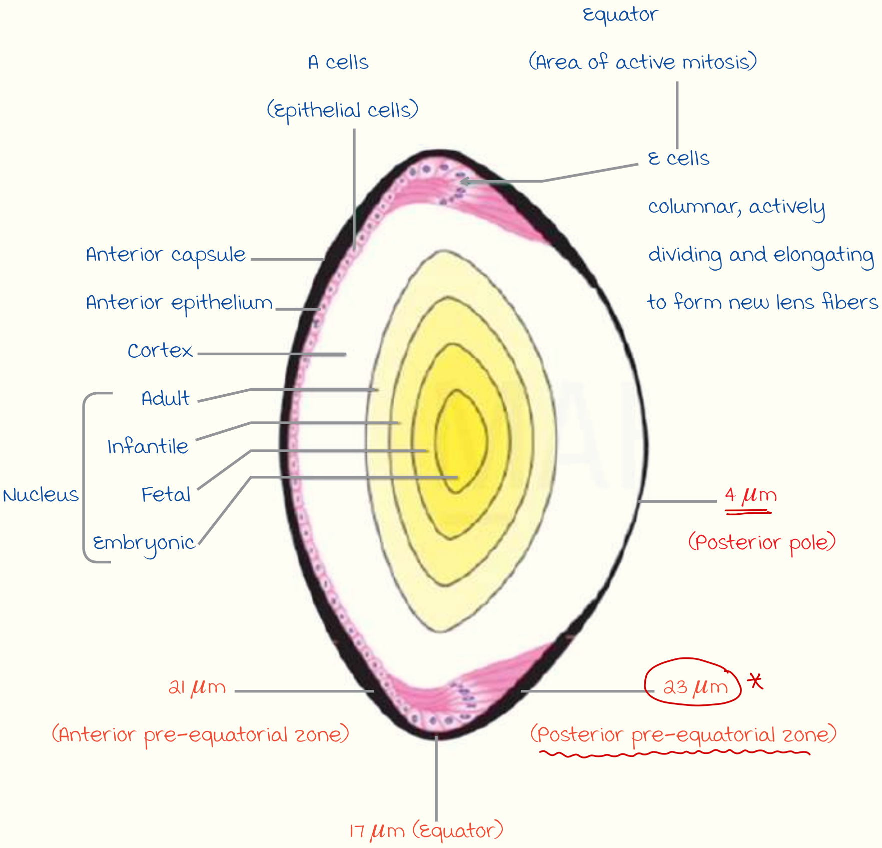
Berger's space / Retrolental space : Space between the lens and vitreous.

Cloquet's canal :

- Potential space for Hyaloid artery to supply lens during embryogenesis.
- Cloquet's canal and Hyaloid artery disappear by 8 months of gestation. If they persist, can lead to :
  1. Mittendorf dots : Remnant of Hyaloid artery on the posterior surface of the lens.
  2. Bergmeister's papillae : Remnant at the optic disc.

Layers of lens

00:18:25



1. Outer most layer : Capsule
   - Capsule is the thickest basement membrane in the body : * Type IV collagen. *
   - Thinnest at : Posterior pole.
   - Thickest at : Posterior pre-Equatorial zone .

2. Anterior epithelium : Singe layer of Cuboidal ep.
   - Have 2 cells :
     1. A cells : metabolism
     2. E cells : Form new lens fibres as they are capable of mitosis. → * ← @ Equator
   - Posterior epithelium is absent as during embryogenesis, posterior epithelial cells elongate to form Primary lens fibres.

3. Cortex
   - Young lens fibres : Outer cortex. *

4. Nucleus :
   - 4 zones :
     - i. E : Embryonic center – Primary lens fibres. ← Oldest lens fibres.

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ii. F : Fetal
iii. I : Infantile
iv. A : Adult
} Secondary lens fibres.

Both Cortex and Nucleus contain lens fibres.

Sutures in lens 00:31:49

Formed during fetal period.

Anterior suture : Y shaped.

Posterior suture : Inverted Y shaped.

Proteins in lens 00:34:45

- water soluble (Crystallins)
  - → α : largest cystallins
    Heat shock proteins.
    ↓
    Prevent denaturatn of other Proteins
  - β : Major portion (55%)
  - → γ
- water insoluble
  - Urea soluble
    - vimentin
    - Filensin
    - phakinin

    ↓
    - Cytoskeletal proteins
    - Their disruption ⇒ Cataract formation
  - Urea insoluble

    MIP-26
    or
    Aquaporin 0

    ↓
    Help to maintain transparency of lens,

HMW (High molecular Weight) proteins :

- HM1 } Water soluble
- HM2
- HM3 } Water insoluble
- HM4 Increase in concentration in cataract. ★AIIMS 15

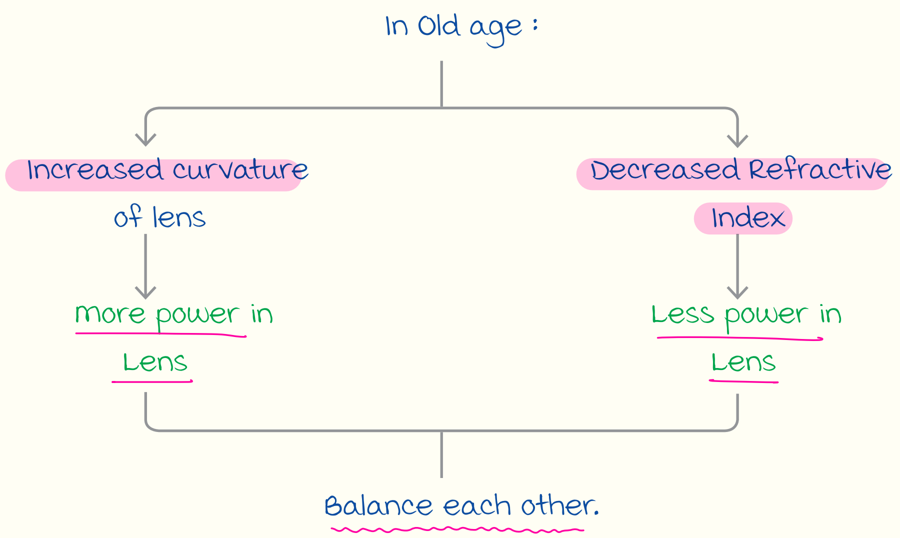
HM4 alone shows increased concentration in Nuclear cataract.

Antioxidant mechanisms in lens and Lens paradox 00:42:42

1. Superoxide dismutase
2. Catalase
3. Glutathione reductase
4. Vitamin C
5. Vitamin E
6. Low oxygen levels around lens :
   - ☆ In vitrectomy, low oxygen level is lost. This leads to increased incidence of cataract formation.

Vitamin A is not an antioxidantin lens.

Lens paradox :



Metabolism in lens 00:48:55

Glucose diffuse insidelens from Aqueous (mainly) and Vitreous.

This glucose is metabolised by :

- Anaerobic glycolysis : > 80% *
- Krebs cycle or HMP shunt : around 15%
- Sorbitol pathway : <5%

Sorbitol pathway is active in :

- Diabetes, Sugar cataract
- Galactosemia

Glucose $\xrightarrow{\text{Aldose Reductase}}$ Sorbitol
(cannot diffuse out
of the lens)

↓

Accumulates in lens.

↓

Increased osmotic
pressure in lens. (Sugar)

↓

⇒ Fluid is drawn inside lens

↓

Opacification, known as Cataract.

Q) Transport of ascorbic acid in lens : Via Sodium dependant SVCT2
(Active process).

LENS: ACQUIRED CATARACT

Introduction

00:00:30

Any opacity in lens is called cataract

mcc of gradual painless loss of vision : cataract

⇒ mcc of blindness in India: cataract

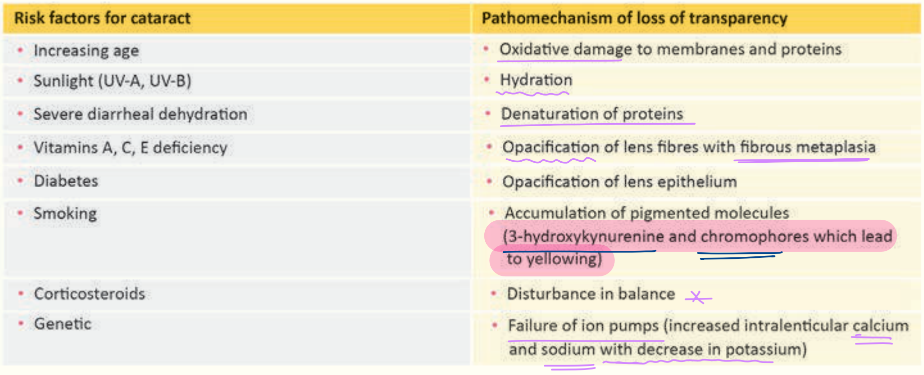
Blindness

Program – NPC BVI (National Programme for Control of Blindness and Visual Impairment).

Definition – visual acuity < 3/60 in the better eye with best possible correction OR visual field < 10 degree.*

Risk factors of cataract

00:06:06



Risk factors for cataract	Pathomechanism of loss of transparency
Increasing age	Oxidative damage to membranes and proteins
Sunlight (UV-A, UV-B)	Hydration
Severe diarrheal dehydration	Denaturation of proteins
Vitamins A, C, E deficiency	Opacification of lens fibres with fibrous metaplasia
Diabetes	Opacification of lens epithelium
Smoking	Accumulation of pigmented molecules (3-hydroxykynurenine and chromophores which lead to yellowing)
Corticosteroids	Disturbance in balance *
Genetic	Failure of ion pumps (increased intralenticular calcium and sodium with decrease in potassium)

↑Ca^{+2}, Na^{+}
↓ K^{+}

Types of cataract

- Acquired
  - metabolic
  - Traumatic
  - Complicated
  - Senile
- congenital cataract
  - Infantile
  - Developmental

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Metabolic cataract : Diabetes mellitus

00:09:00

Diabetes mellitus causes :

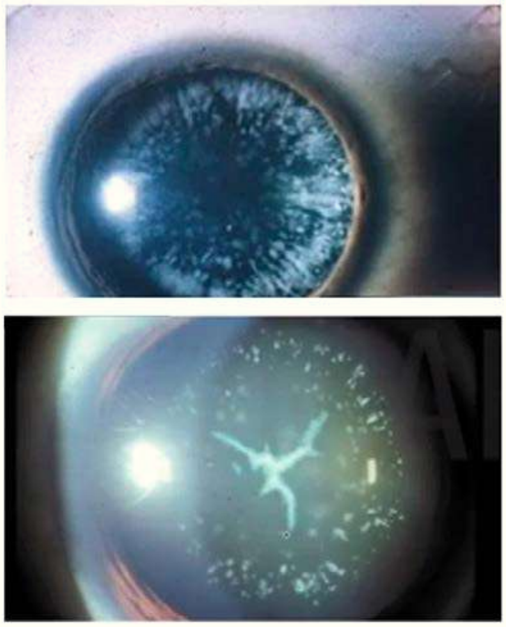
1. Snow fake cataract (cortical cataract)

   MC in type I DM - young adults

2. Presenile cataract (senile cataract before 50 years of age)

   it occurs due to increased carbamylation of crystallins

⇒ 3. Fluctuating refractive errors (d/t uncontrolled 'G' levels)



Snowflake Cataract

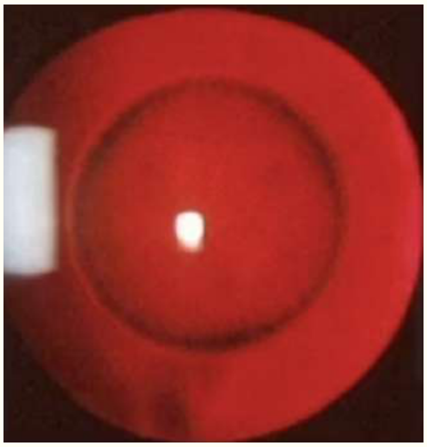
Metabolic cataract : galactosemia

00:14:15

Galactosemia causes :

Oil droplet cataract (posterior subcapsular cataract)

This is the only reversible cataract



Galactosemia - Oil Droplet Cataract

Galactosemia is an autosomal recessive disorder.

Galactose ——✕——→ glucose
& its metabolite
(Galactitol (Dulcitol)
↓
Cataract

- Gal-1-PUT
- UDP Galactose 4 epimerase
- Galactokinase

↓
Galactosemia c̄ Galactokinase def
⇒ Only Cataract

def : MR
Hepatomegaly
FTT

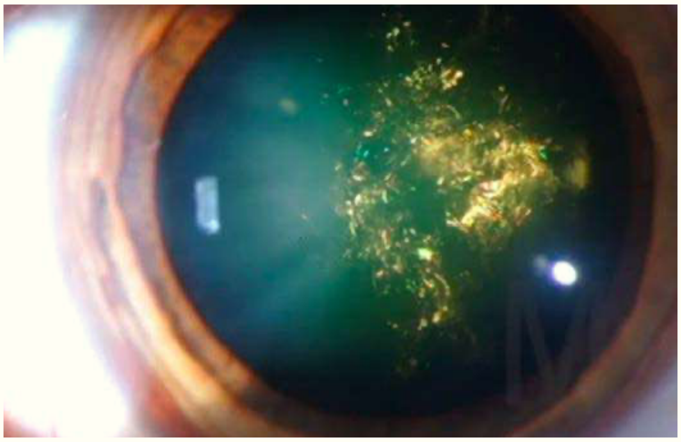
Q) Infant : B/L cataract : presumed to be Galactosemia : Investigation to confirm : Urine Assay - measure reducing substances in urine

Metabolic cataract : Myotonic dystrophy

00:22:11

It causes :

1. Christmas tree cataract (posterior subcapsular cataract)
2. ✓ Presenile cataract



Myotonic Dystrophy - Christmas Tree Cataract

Metabolic cataract : Wilson's disease, Parathyroid tetany

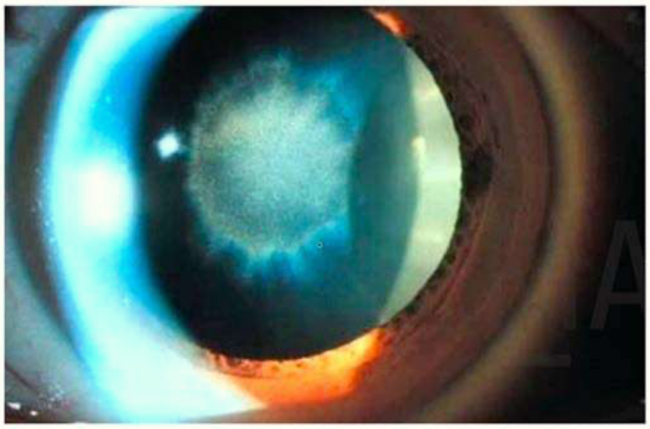
00:24:09

It causes:

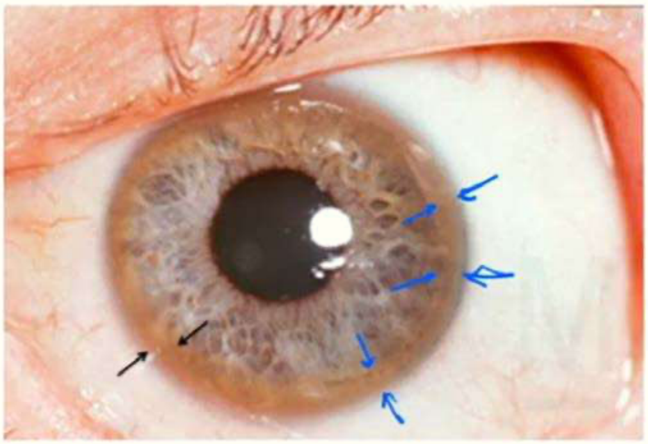
sunflower cataract – due to the deposition of cuprous oxide and it can affect multiple sites of the lens. It is stellate shaped.

KFD

Kayserfleischer ring – due to the deposition of copper in descemet's membrane of cornea (periphery of cornea) *



Wilson's Disease – sunflower Cataract



Kayser Fleischer Ring

Diagnosis

*

IOC – 24hour urinary copper levels measurement

Parathyroid tetany

It leads to hypocalcaemia, which causes anterior and posterior subcapsular cataract or lamellar cataract in children.

↓ Aquired

↓ Cong.

Active space

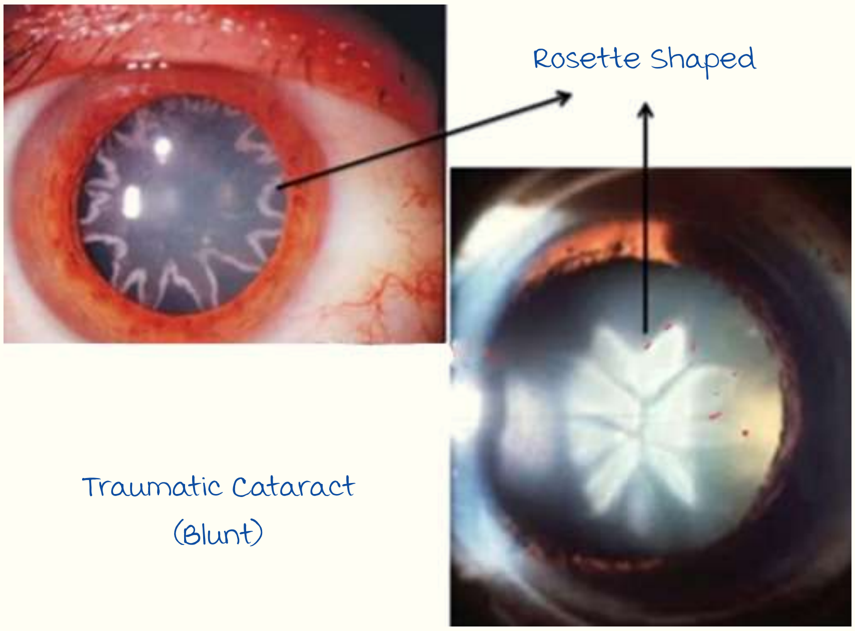
Traumatic cataract

00:30:25

1. Blunt trauma causes – rosette shaped cataract (posterior subcapsular cataract).

   → heat

2. Infrared rays causes– glassblower's cataract, occurs due to true exfoliation of lens capsule.
3. Lightning/ electric shock – anterior capsular opacities.



Traumatic Cataract
(Blunt)

Complicated cataract

00:35:00

The causes are :

→ 1. Uveitis – MCC
2. myopia
3. Angle closure glaucoma
4. Retinitis pigmentosa

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

Appearance – bread crumb appearance and on showing light, polychromatic lustre is noted. ✓

Site – posterior subcapsular cataract.

Senile Cataract: Nuclear cataract pathology 00:43:44

It is also known as age related cataract. It occurs around 50 - 70 years of age.

There are four types :
Nuclear cataract
A/K/A central cataract
C/f - loss of vision in daytime
Improvement of vision in dim light /night

Pathology - it occurs due to sclerosis, it causes hard cataract
↓
Increase in refractive index of lens
↓
Increase in power (converging of lens)
↓
→ Index myopia - loss of far vision or near sightedness
leads to second sight

Second sight - improvement of near vision due to nuclear cataract in a patient who is already presbyopic.

Senile Cataract: Nuclear cataract colour 00:51:15

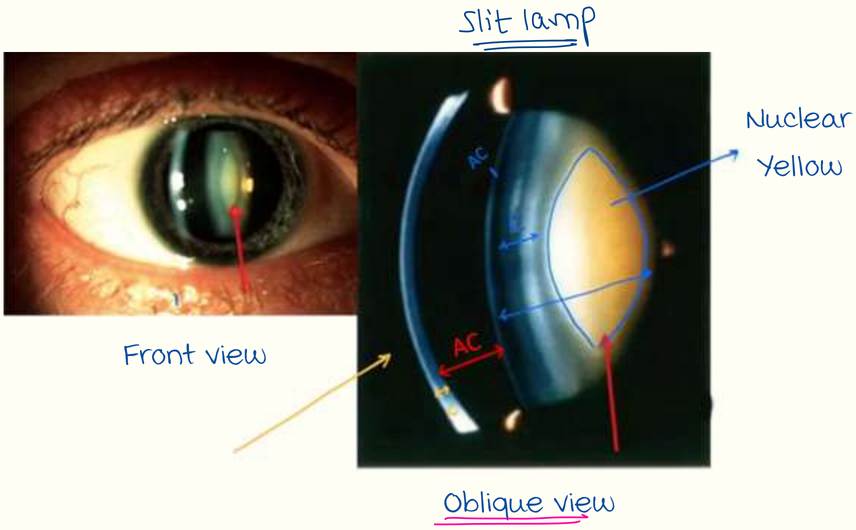
Colour

grade 1 - yellow due to deposition of urochrome pigment,
c/f yellow coloured vision called xanthopsia *

grade 2 - Amber (orange)

grade 3 - Brown called as cataractabrunescens

grade 4 - Black called as cataracta nigra



Nuclear Cataract

Senile cataract : Cortical cataract

00:57:00

It is A/K/A peripheral cataract.

Loss of vision in dim light/night.

Pathology – hydration of lens causes soft cataract

The earliest change – formation of water clefts and vacuoles

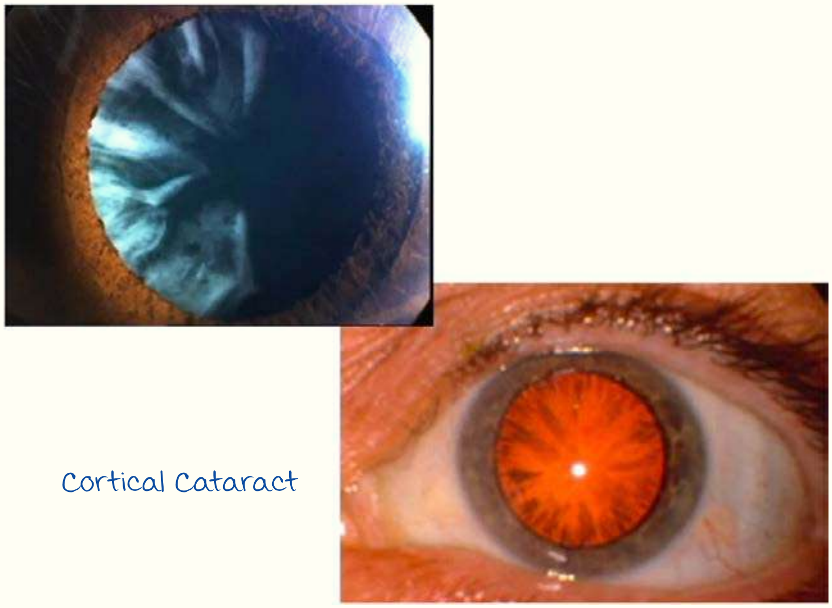
↓

lamellar separation

↓

Wedge shaped opacities are seen – cuneiform cataract

– mc seen inferonasally



Cortical Cataract

Senile cataract : Immature, mature, hypermature cataract

1:03:18

The cuneiform cataract progresses to immature cataract.

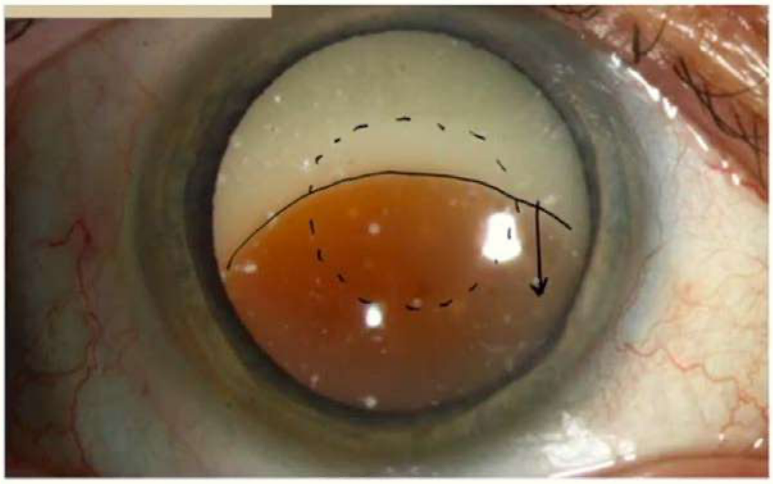
It is seen as dirty greyish colour.
Iris shadows is seen over lens surface.
As the fluid accumulates it causes swelling of the lens and immature cataract progresses to intumescent cataract (swelling). It is also called a mature cataract. It is chalky white in colour and no iris shadow.

mature cataract progresses to hypermature cataract.

It can present as

- morgagnian
  - Liquefaction of cortex
  - ↓
  - leads to sinking of nucleus
- sclerotic
  - anterior capsule becomes wrinkled, leathery

mc compilation of hypermature cataract is subluxation of lens. ✓
mc compilation of morgagnian cataract is phacolytic glaucoma.



morgagnian Cataract

Posterior subcapsular cataract : Cupilliform Cataract 1:13:31

⇒ Causes max. visual loss
↓
as opacity is present axially @ nodal point of eye

C/o : - More LOV for near
vision improves in dim light
· GLARE (Characteristic) — Inability to drive @ night

Path: d/t dysplasia of 'E' cells ⟶ posterior migration & Abberant enlargement
↓
form plaque K/A Bladder cells or Wedl cells

Other causes of posterior subcapsular cataract :

- Corticosteroids (systemic steroids)
- X rays
- C Chloroquine
- B Busulfan
- N Neurofibromatosis- 2

· Galactosemia
· Myotonic dystrophy
· Blunt trauma
· Complicated cataract

G - Glaucoma
↑
T - Topical Steroids
C - Cataract
↑
S - Systemic Steroids

Senile:after cataract subcapsular cataract 1:23:19

Other causes of anterior subcapsular cataract :

AACG

1. Atopic dermatitis: shield cataract
2. Amiodarone
3. Chlorpromazine
4. Gold deposits
5. Acute angle closure glaucoma (AACG)

Active space

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Active space

LENS - CATARACT SURGERIES

Treatment of acquired cataract 00:00:06

1. Intra-Capsular Cataract Extraction (ICCE) :
   - Not done now.
   - Only current indication : Subluxation of lens due to >180° zonular dehiscence.
   - Complete lens with the capsule is removed.

2. Extra-Capsular Cataract Extraction (ECCE) :
   - Lens is removed but not the capsule.
   - Techniques :

	Site of Incision	Size of Incision	
a. Conventional ECCE	Limbus	8 – 10 mm (equatorial d = 10mm)	9
b. Small Incision Cataract Surgery (SICS)	Sclera	5 – 7 mm	6
c. Phacoemulsification	Cornea	2.2 – 3.2 mm	3

d. Femtosecond Laser Assisted Cataract Surgery (FLACS) : Latest surgery
- Laser used- Nd : glass (1053 nm)
- mechanism of action : Photodisruption
- → Time duration of each laser burn : 10^{-15}s

Burn : Ablation
Coag : Coagulation
Break : disruption

Steps of Phacoemulsification 00:13:09

Pre-operative surgical preparation :
- Dilate the pupil : Tropicamide with Phenylephrine.
- Anesthesia – Topical : Proparacaine.
  Local : Peribulbar (preferred).
  Retrobulbar.

Peribulbar injection : Bupivacaine : long acting
+
Lignocaine : fast onset
+

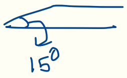
Active space

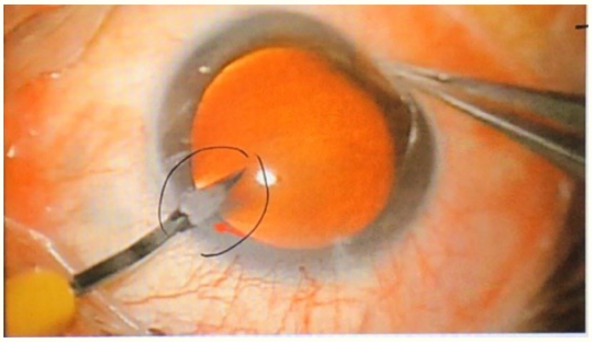
1:200000 Adrenaline
+
Hyaluronidase ✓

- Povidone iodine / Betadine solution instilled in the conjunctival SAC.

Intraoperative steps :

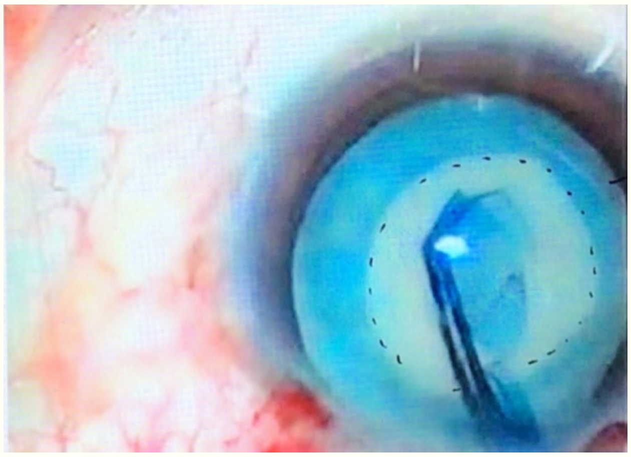
1. Paracentesis (Sideport creation) :
   - Given using 15° blade or MVR blade.





a. Perform staining of anterior capsule by Trypan Blue dye. *

b. Continuous Curvilinear Capsulorrhexis (CCC) :



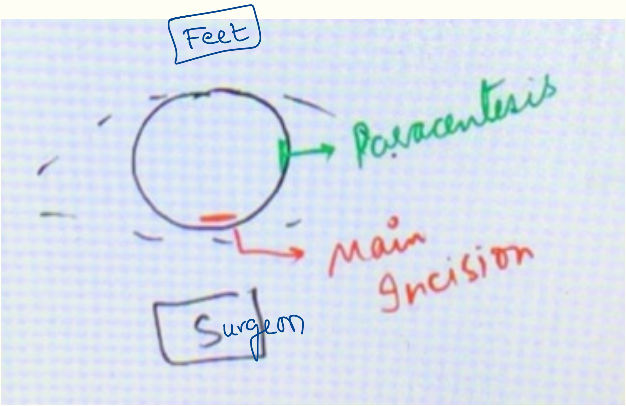
Active space

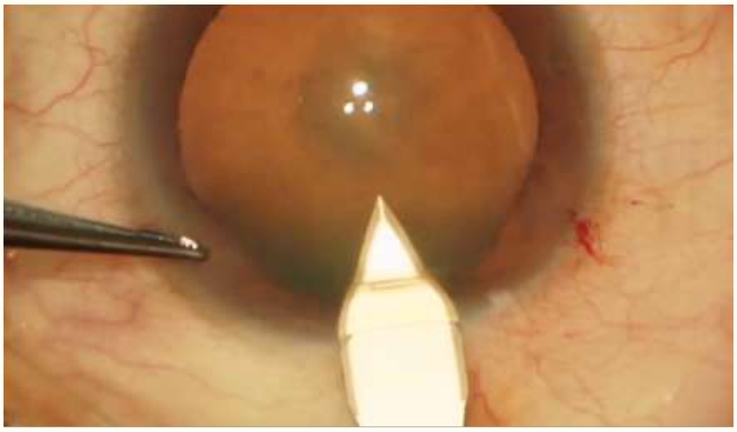
- Anterior capsule : Blue colour
- Center : CCC

Intraoperative steps of Phacoemulsification

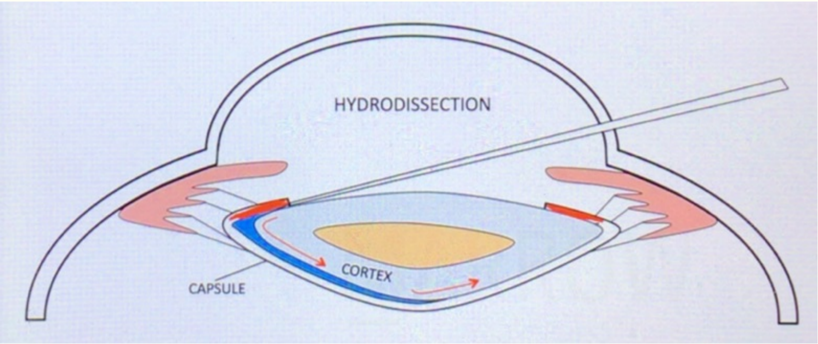
00:17:30

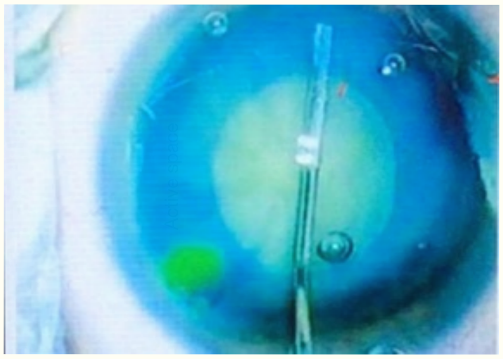
2. main port incision made using a Keratome at 3 O'clock hours away from paracentesis. *





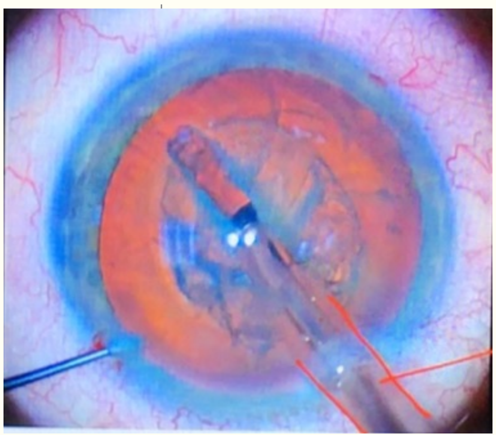
a. Hydrodissection : A jet of fluid is used to separate the posterior capsule from the cortex.





b. Hydrodelineation : Jet of fluid
↓
seperate Endonucleus from Epinucleus.

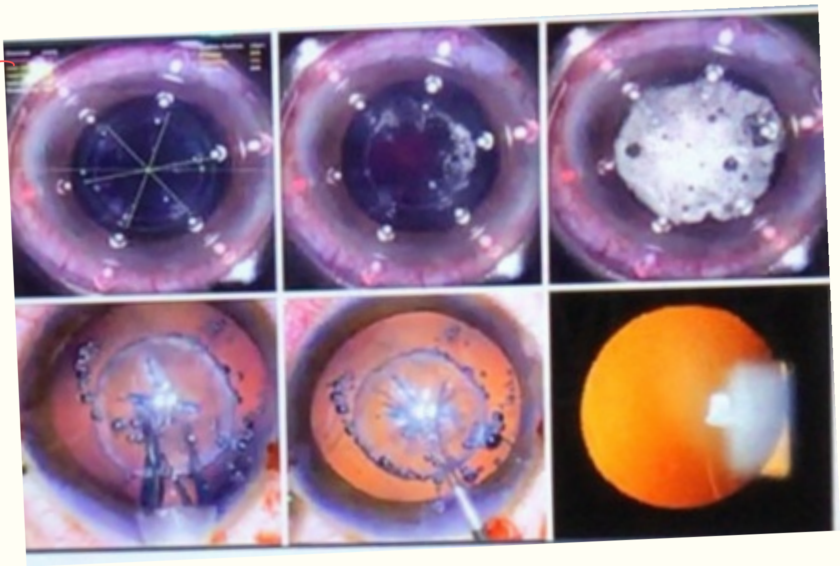
c. Emulsification of Nucleus and Nuclear fragmentation:



- using phacoprobe
↓
Piezoelectric crystal
↓
electrical E → mech. to & fro movements

↓
Left with empty capsular bag
↓
d. Implant Intra ocular lens

FLACS



Nuclear fragments

Laser performs
- CCC
- Nuclear fragments
- Marks main Incision

Active space

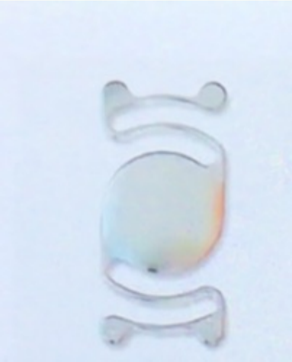
Intra ocular lens

00:36:30

Best site for IOL implantation:In the capsular bag.

Types :

- ACIOL : made of PMMA.



S – Shaped Haptic.

Active space

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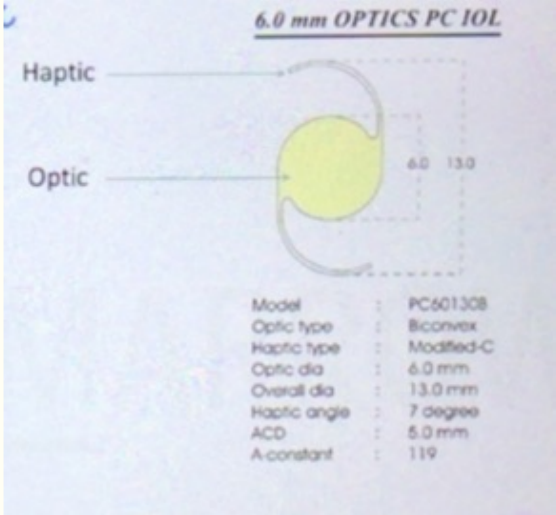
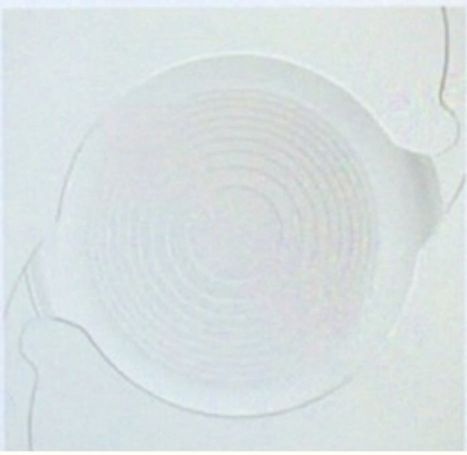
- PCIOL : made of Silicone or Acrylic.



Model	:	PC601308
Optic type	:	Biconvex
Haptic type	:	Modified-C
Optic dia	:	6.0 mm
Overall dia	:	13.0 mm
Haptic angle	:	7 degree
ACD	:	5.0 mm
A-constant	:	119

C – Shaped Haptic.

- multifocal IOL :



Zones appear as rings.

Drawbacks :

a. Patient experiences glares.*

b. Patient complaints of halos.*

Toric IOL : In pre-existing astigmatism.

- Square edged IOL : To reduce occurrence of Posterior capsular opacification (PCO).

Active space

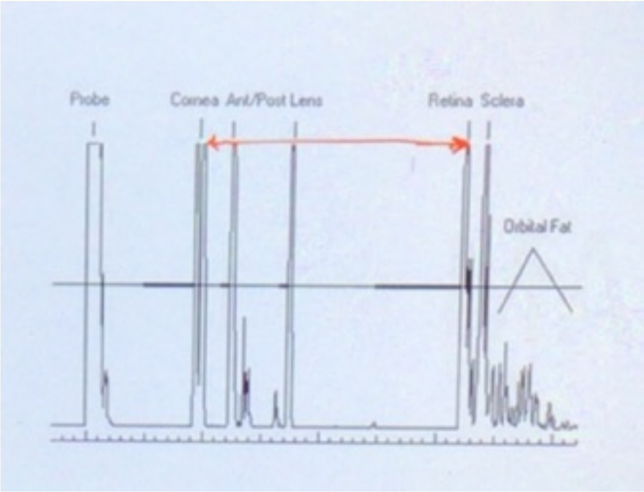
Calculation of IOL power

00:44:07

Calculation of IOL power is known as Biometry.

Steps :

1. measure corneal curvature : Keratometry. ✓
2. measure the axial length of the eye (Anteroposterior diameter) : A scan ultrasonography.
    - A : Amplitude
    - Normal axial length : 24mm
    - ⇒ Linear Distance between corneal echo and retina echo is calculated = Axial length.



3. Formulae :
    a. SRK-T
    b. Holladay : If axial length > 24.5 mm
    c. Hoffer Q : If axial length < 22 mm
    d. Haigis-L : Post LASIK patient(change in corneal curvature).

taking into account :
: pre Lasix measurements
post Lasix

Intra-operative complications of cataract surgery

00:50:51

a. Posterior capsular rupture (PCR)
- PCIOL in the bag is not implanted .
- ACIOL or PCIOL (in the sulcus) is used instead.
- PCIOL (in the sulcus) uses multipiece IOL or 3 piece IOL.

↓ B/w Iris & capsule

Active space

b. UGH Syndrome (Uveitis, Glaucoma, Hyphema)

- Due to ACIOL causing iris chaffing. (Iris :moving → Constantly rubbing on IOL)
- Hyphema : Collection of blood in the anterior chamber.
- Treatment : Explant ACIOL.
  Implant PCIOL. ✓

c. Expulsive Choroidal hemorrhage :

- Occurs due to sudden changes in IOP. *
- * Source of hemorrhage : Short posterior Ciliary artery. *

d. Descemet's membrane detachment :

- Treatment : Descemetopexy * (air bubble in anterior chamber).

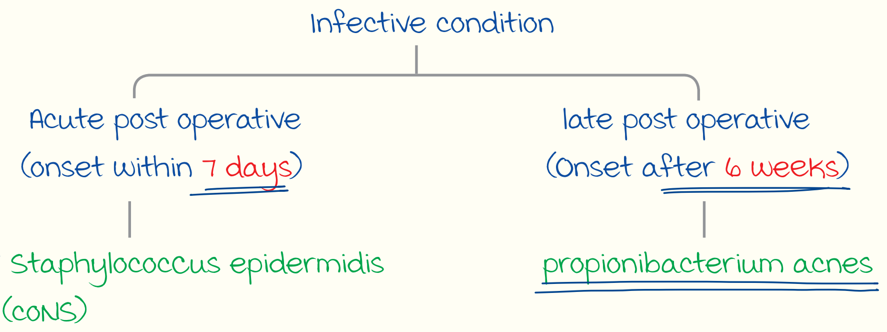
Acute post-operative complications of cataract surgery 01:01:50

a. Flat /Shallow anterior chamber :

- Causes :
  1. Leaking incisions
  2. Pupillary block glaucoma
  3. Ciliochoroidal detachment

b. Endophthalmitis : most dangerous Compl^n

- Suppurative inflammation of all layers of eye ball except Sclera.



Infective condition

Acute post operative (onset within 7 days) ⇒ Staphylococcus epidermidis (CoNS)

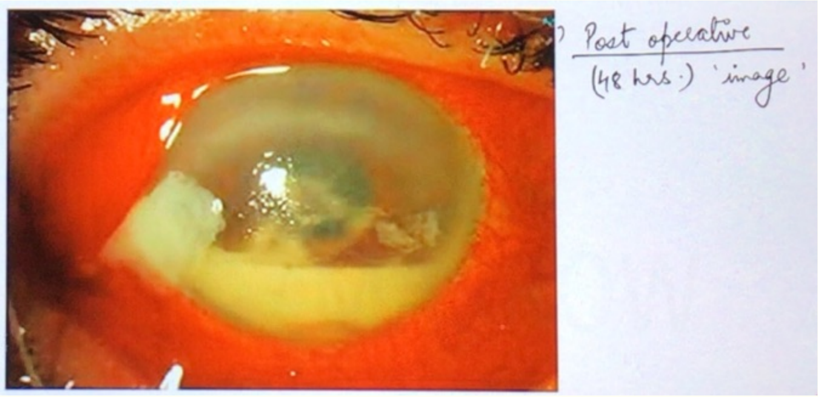
late post operative (Onset after 6 weeks) propionibacterium acnes

Active space

Clinical features : Pain + Redness + Loss of vision
Congestion *
Corneal haze (corneal inflammation)

Hypopyon – Collection of pus in anterior chamber.

Vitritis – Cells, yellowish exudates in vitreous.



Acute onset Endophthalmitis.

- Treatment :

Vision

Blind (no perception of light)

treatment of choice : Evisceration

⇒ • Remalal of all layers of eyeball, except sclera (≡ def of Endopthalmitis)

Vision : present (whatever)

⇒ • Intravitreal Antibiotics : for post - seg
- vancomycin (1mg / 0.1ml)

+

- ceftazidime (2.25 mg / 0.1 ml)

+

- Topical antibiotics : for Ant segment
  - broad spectrum

+

- Pars plana vitrectomy

No use of Systemic antibiotics. (local d(s))

- Other types of Endophthalmitis and causative organisms :
  1. Post – traumatic endophthalmitis – Bacillus cereus
  2. Endogenous endophthalmitis – Klebsiella
  3. Endogenous fungal endophthalmitis – Candida

Active space

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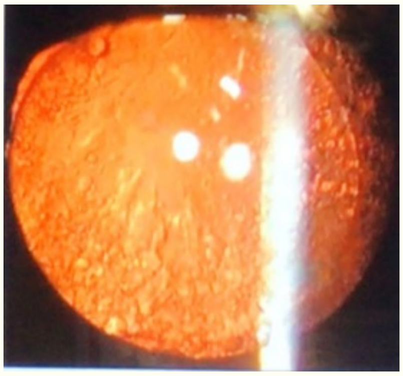
Late post-operative complications

01:19:32

a. Posterior capsular opacification (PCO)

- It is also known as After cataract / 2° cataract.
- The most common late complication (6-12 months after surgery).
- Types :

1. Elschnig pearls (90%): Forms due to posterior migration of residual epithelial cells.



2) Sommering's Ring : 10%

Rx PCO :

Nd-yag laser posterior Capsulotomy
(1064nm)
- Photodisruption (break)

b. Displacement of IOL :

- Sunrise syndrome : Superior IOL d/L
- Sunset syndrome : Inferior d/L of IOL
- Lost Lens " : Complete subluxation

c. Irwin Gass Sx : usually 6-10wks After Sx.

- Cystoid macular edema
- Bullous Keratopathy
- Vitreous touch

→ triad

d. Anterior capsular Phimosis :

Contraction of Ant. capsule d/t fibrosis

LENS: CONGENITAL CATARACT, ECTOPIA LENTIS AND MISCELLANEOUS

Congenital cataract

00:00:05

Types

Infantile
- visible at birth oR before 1 year of age
- ⇒ severe loss of vision (as it interferes with development of faveal fixation)

Usally occurs by 3-4 months of age

· Lamellar | Zonular

Developmental
- visible after 1 year of age

⇒ minimal loss of vision

· Blue dot | Punctate

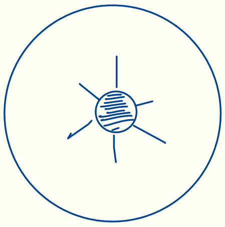
Types of Congenital cataract

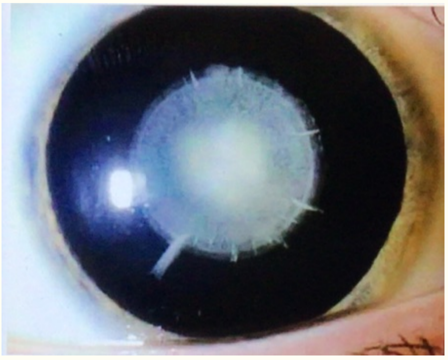
00:05:08

1. Blue dot cataract / Punctate cataract
   - ⇒ most common congenital cataract.
   - most common developmental cataract.
   - Associated with downs syndrome.

2. Lamellar cataract / Zonular cataract
   - most common infantile cataract.
   - Associated with :
     a. Congenital rubella syndrome (CRS)
        most common cataract in CRS is Nuclear pearly cataract.
     b. Vitamin D deficiency
     c. Autosomal dominant inheritance
   - Clinical features :
     1. Opacity affects fetal nucleus.
     2. Spoke like opacities seen known as Riders.

Active space

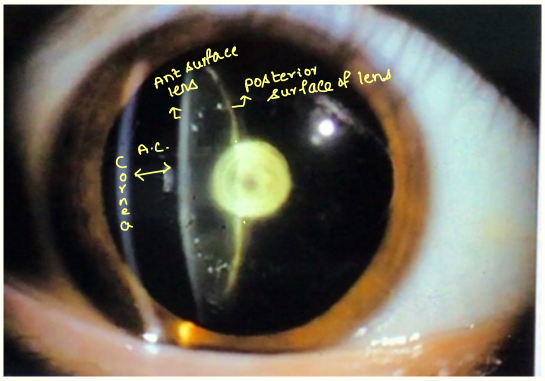




3. Posterior polar cataract

- Onion ring or whorled appearance.

Slit lamp



- Associated with :
  a. mittendorf dots
  b. Persistent hyperplastic primary vitreous (PHPV)
  c. PITX3 gene mutation

Treatment of Congenital cataract

00:14:16

Treatment : Surgery

Lens aspiration (child - lens soft : i.e can be aspirated instead of breaking)
+
Posterior capsulectomy (to reduce occurrence of PCO) (central cut)
+
Anterior vitrectomy (To prevent mixing c̄ Aqueous)
+
IOL implantation (Usually done after 2 years of age) (as eye ball size - Grows)

ECCE surgery

ECCE surgery

Active space

IOL power in children :

- Child <2 years : under correct by 20 %
- Child 2 - 8 years : under correct by 10 %

Timing of surgery for congenital cataract :

- Unilateral, dense : Immediately (before 4-6 weeks of age), otherwise sensory deprivation amblyopia may occur.
- Unilateral, partial : Anti-amblyopia occlusion therapy.
- Bilateral, dense : Surgery by 12 weeks.
- Bilateral, partial : Defer till 5 years*

Ectopia lentis

00:22:53

It refers to displacement of lens.

Causes :

1. Most common : Blunt trauma
2. Hypermature cataract
3. Aniridia (WAGR)
4. Familial : Autosomal dominant
5. Marfan syndrome :
   - Direction of Ectopia lentis : Superotemporal.
   - Other ocular clinical features: Megalocornea

- Cornea plana – char. of Marfan Sx
- Hypoplasia of dilator pupillae muscle
- Axial myopia
- Lattice degeneration of retina

6. Homocystinuria
   - Direction of Ectopia lentis : Inferonasal
   - Deficiency of Cysteine leads to zonule break, leading to loss of accommodation.

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow masterclasses

7. Weill Marchesani syndrome
   - Direction of Ectopia lentis : Anterior / Forward
   - Usually the patients have : Short stature Stubby fingers (W)

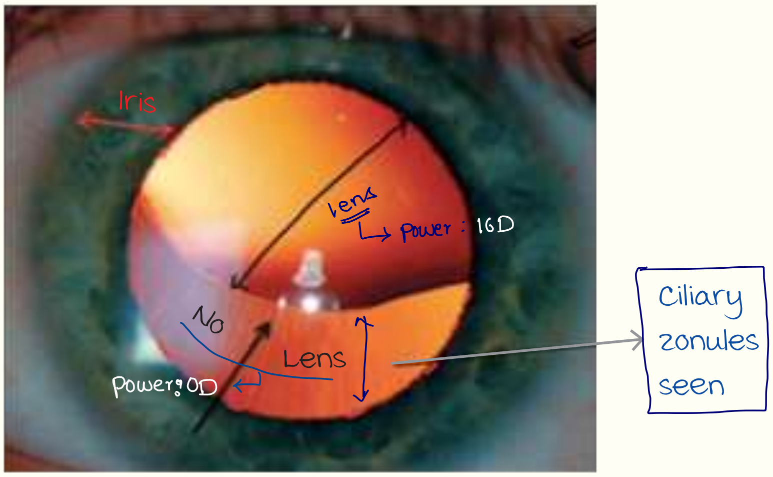
$\bar{c}$ microspherophakia

8. Hyperlysinemia
9. Sulfite oxidase deficiency
10. Ehlers-Danlos syndrome

Ectopia lentis: Clinical presentation

00:31:40

Superior Ectopia lentis :



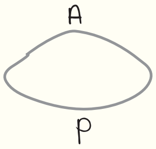
- Sign : In the pupillary area, Golden crescent is seen.
- Symptom : Diplopia (mono-ocular)

Lenticonus

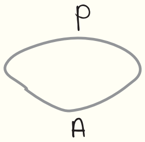
00:35:35

It refers to cone shaped lens.

Anterior lenticonus

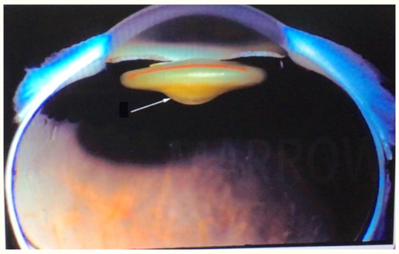


posterior lenticonus



S - Spina Bifida
A - Alport Sx
W - Waardenburg Sx

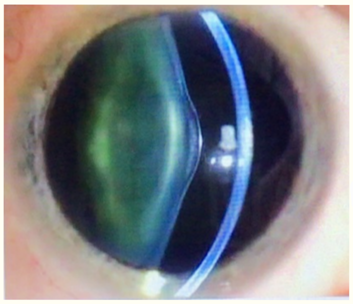
Active space



Posterior lenticonus

. LOWE Sx
↓
Other ocular features:

i) Cong. cataract
ii) Cong. Glaucoma
iii) Microphakia
iv) Posterior LentiConus



Anterior lenticonus

Microspherophakia

00:39:22

Characterised by small, spherical lens.

The increased curvature of the spherical lens leads to myopia.

Due to the small size of the lens, it can dislocate into the anterior chamber. This can lead to Secondary angle closure glaucoma.

The equator is visible in pupillary area.

It is usually associated with Weill-marchesani syndrome.

Active space

Active space

GLAUCOMA - BASICS AND INVESTIGATIONS

Definition 00:01:30

OTP

1. Progressive , characteristic optic disc changes (Tested by : Ophthalmoscopy)

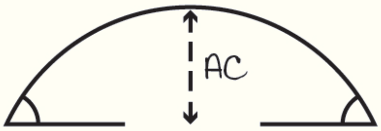
⇒ 2. Irreversible visual field changes (Tested by : Perimetry)

3. Intraocular pressure[IOP] may or may not be raised (Tested by : Tonometry)

Flow of Aqueous 00:05:17

- Site of production Non Pigmented epithelium of ciliary processes.
  - 70%- Active secretion.
  - 20%- Ultrafiltration.
  - 10%- Osmosis.
- "Production → Secreted into posterior chamber → To Anterior Chamber via Pupil"

↓



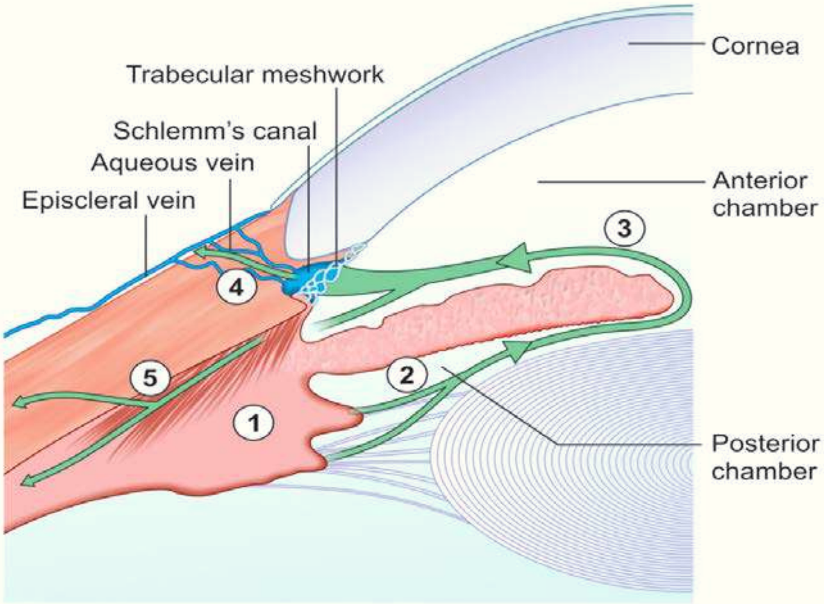
Angle of Anterior Chamber (Iridocorneal Angle)

Outflow

Trabecular (90%)
Trabecular meshwork
↓
Schlemn's Canal
↓
Aqueous Vein
↓
Episcleral Vein
↓
Drains into Cavernous sinus via Ophthalmic Vein

Uveosacral (10%)
drains into Supraciliary space
(space between sclera and Ciliary body)

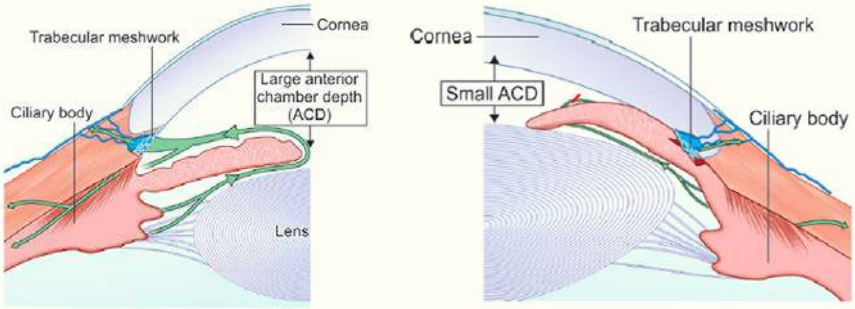
Active space



1. Ciliary Process
2. Space between iris and posterior chamber
3. Anterior chamber
4. Iridocorneal Angle
5. Space between sclera and Ciliary body

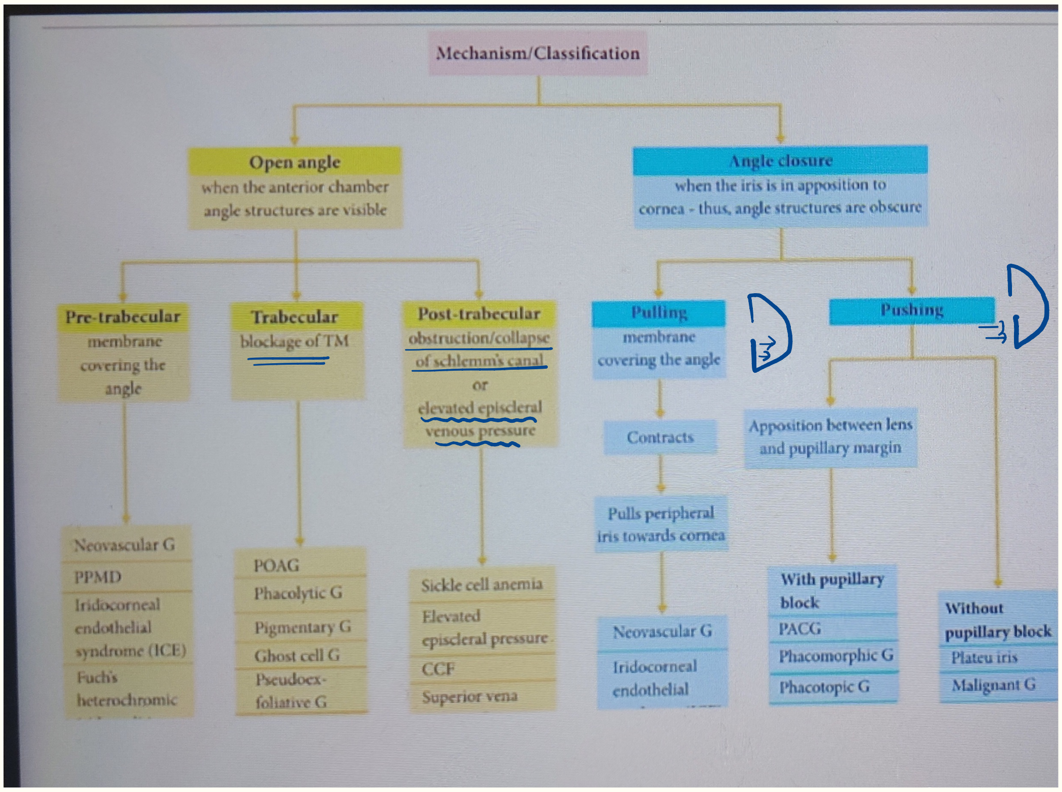
Types of Glaucoma

00:15:00



Open Angle Glaucoma
Path: Trabecular meshwork
Treatment: Trabeculectomy

Angle Closure Glaucoma
Path: Iris
Treatment: Iridectomy


Mechanism/Classification
Open angle
when the anterior chamber angle structures are visible
Angle closure
when the iris is in apposition to cornea - thus, angle structures are obscure
Pre-trabecular
membrane covering the angle
Trabecular
blockage of TM
Post-trabecular
obstruction/collapse of schlemm's canal
or
elevated episcleral venous pressure
Pulling
membrane covering the angle
Pushing
Contracts
Pulls peripheral iris towards cornea
Apposition between lens and pupillary margin
Neovascular G
PPMD
Iridocorneal endothelial syndrome (ICE)
Fuch's heterochromic
POAG
Phacolytic G
Pigmentary G
Ghost cell G
Pseudoex-foliative G
Sickle cell anemia
Elevated episcleral pressure
CCF
Superior vena
Neovascular G
Iridocorneal endothelial
With pupillary block
PACG
Phacomorphic G
Phacotopic G
Without pupillary block
Plateu iris
Malignant G

Pushing and pulling mechanism.

Active space

Investigations

00:21:43

A. Tonometry- (measurement of IOP)

- Normal IOP- 11 to 21 mm Hg (16±5)
- IOP has a diurnal variation(max in late morning from 8am to 12noon)

Types of Tonometry :

1. Digital Tonometry [DT]

- IOP low : Soft.
- IOP high : Rocky Hard.
- IOP normal : Firm.
  - DT is the techniques of choice in patients wearing Keratoprosthesis

2) Indentation Tonometry

Schiotz Tonometer

- High displacement Tonometry
- Pt - supine
- Footplate : on cornea

Rebound Tonometer

- Choice for self monitoring of Iop.

Active space

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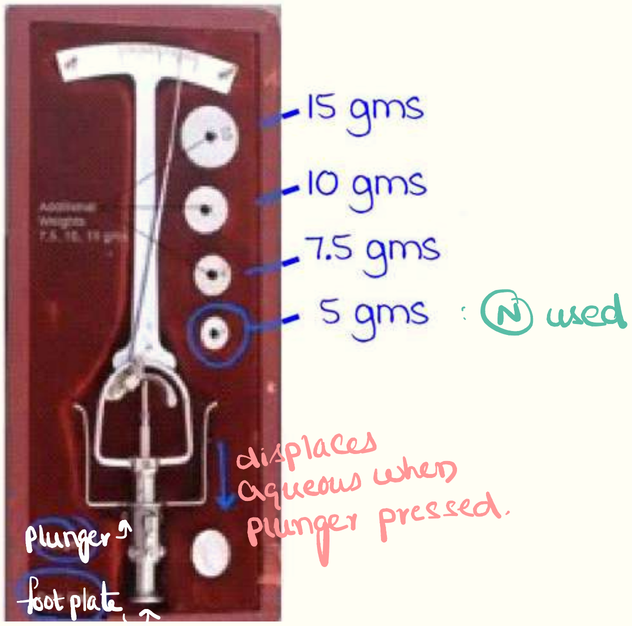
Schiotz Tonometer


Additional Weights 7.5, 10, 15 gms
15 gms
10 gms
7.5 gms
5 gms : Ⓝ used
displaces aqueous when plunger pressed.
Plunger
foot plate

Reading shown on Schiotz tonometer are converted into IOP using Friendwald's Nomogram.

- not a good tonometer because it has interobserver and Intraobserver variability.

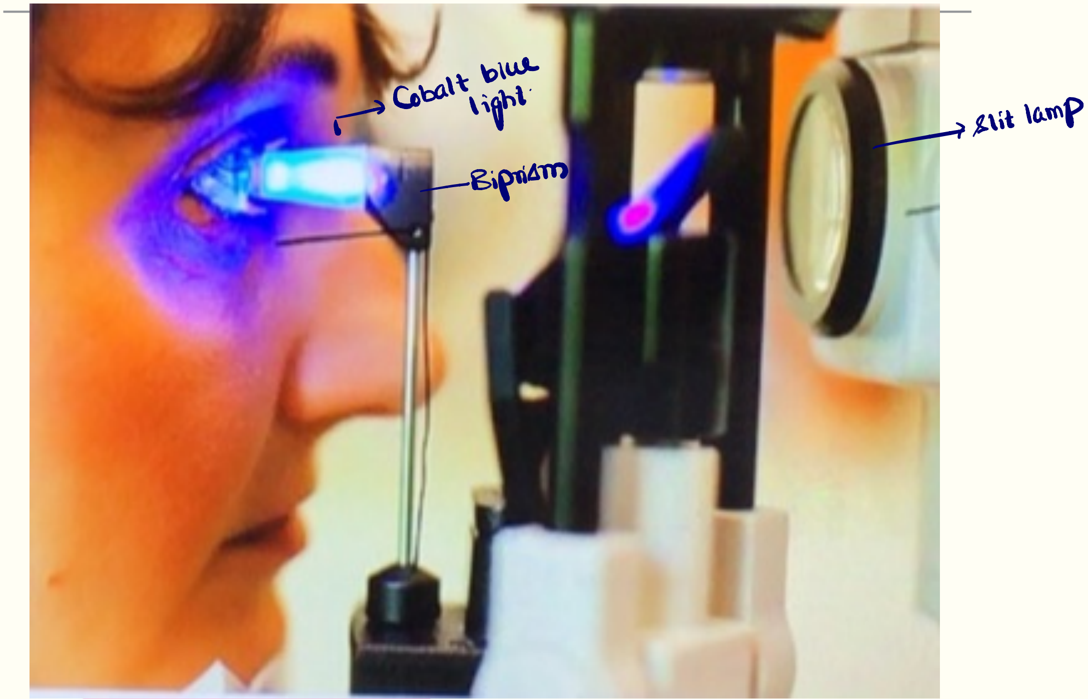
3. Applanation Tonometry[AT]
   - Based on the "Imbert-Fick principle"
     (i.e. $P=F/A$)

Fixed Force AT
E.g. maklakov and Barraquer
– not used

Fixed Area AT
E.g.
i. Goldmann's AT
ii. Perkins AT
iii. mackay marg AT
iv. Pneumatic AT
v. Pascal AT

Goldmann's AT

00:33:47



- Gold standard tonometry
- It is a slit lamp mounted instrument

Looks like an Ostrich

Active space

- method :

Topical Anesthetics (cornea)

↓

Stain cornea with fluorescein dye(strips)

↓

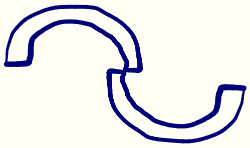
Biprism is moved forward to touch cornea, illuminated under Cobalt Blue Light.

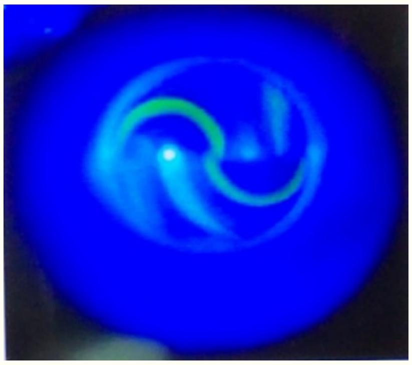
↓

Biprism splits the light into 2 semicircles

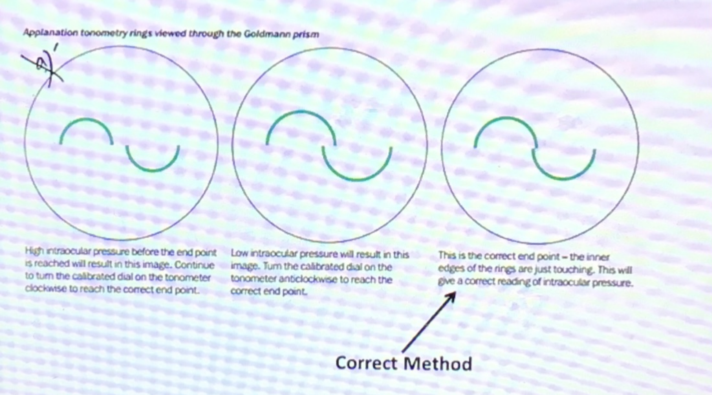
↓

Force adjusted till the inner margins of both just touch each other.





- Applanation area is fixed = 3.06mm* diameter



Applanation tonometry rings viewed through the Goldmann prism

High intraocular pressure before the end point is reached will result in this image. Continue to turn the calibrated dial on the tonometer clockwise to reach the correct end point.

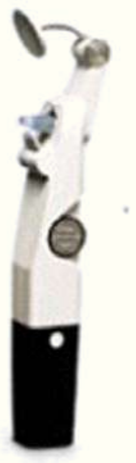
Low intraocular pressure will result in this image. Turn the calibrated dial on the tonometer anticlockwise to reach the correct end point.

This is the correct end point – the inner edges of the rings are just touching. This will give a correct reading of intraocular pressure.

Correct Method

b) Perkins AT

00:41:33



- It is a hand held instrument
- Choice of tonometry in:
    - Children ✓
    - bed ridden ✓
    - In Operation theatre (anaesthetized) ✓

Mackay margAT

- Choice in irregular, edematous cornea.

PneumaticAT

- For continuous IOP measurement
- IOP measurement through contact lens

PascalAT or Dynamic Contour Testing

Active space

Telegram--@latestpgnotes

- not influenced by corneal thickness

↓

Choice for IOP measurement in post LASIK cases (post refractive surgery)

IOP measurement is influenced by corneal thickness. So, in

Requires more force for Applanation ← Thick cornea: measured IOP is False High ($P \propto F$)

Thin cornea: measured IOP is False Low

Non-Contact Tonometry[NCT]

00:47:52

a. Grolman's Tonometry/ Airpuff Tonometry: choice in screening of glaucoma. (ico contact ↓ Risk of infecn)

b. Reichat ocular Response Analyzer[ORA]: Jet of air measures "Corneal Hysteresis" → biomechanics

5. Trans Palpebral Tonometry: "Through eyelid"

  a. Diaton Tonometer

  b. Proview Tonometer

Perimetry

00:52:19

"Visual Field Examination"

↓

Usually measures only the central 30 degree visual field

Types

Kinetic : moving obj. used

e.g. Goldmann's Perimeter

Static

e.g. Humphrey Filed Analyzer (HFA) – clinically used

Active space

Telegram--@latestpgnotes

HFA :

- Bowl type screen
- Testing pattern :
  - 24-2(central 24 degree visual Field is being tested)
  - 30-2(central 30 degree visual Field is being tested)

Testing strategies :

- Supra treshold → Screening
- Treshold → monitoring Glaucoma pts
- SITA : Swedish Interactive Treshold Algorithm
  (mlc)
- Fastpac
- TOP - Tendency oriented perimetry

Newer :

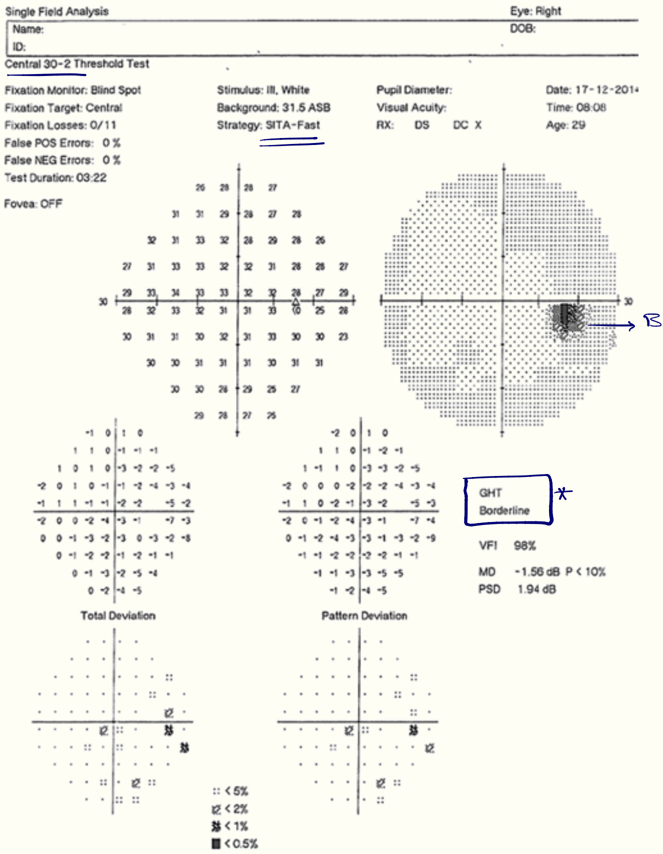
AIIMS ☆ SWAP : Blue on Yellow stimuli
- tests Koniocellular pathway

FDT : Flickering black & white stimuli
Used for screening in Infants / children
tests Magnocellular pathway

Ring : peripheral VF = tested

tests Parvocellular pathway

- HFA output :



Single Field Analysis Eye: Right

Name: DOB:

ID:

Central 30-2 Threshold Test

Fixation Monitor: Blind Spot
Fixation Target: Central
Fixation Losses: 0/11
False POS Errors: 0 %
False NEG Errors: 0 %
Test Duration: 03:22

Fovea: OFF

Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA-Fast

Pupil Diameter:
Visual Acuity:
RX: DS DC X

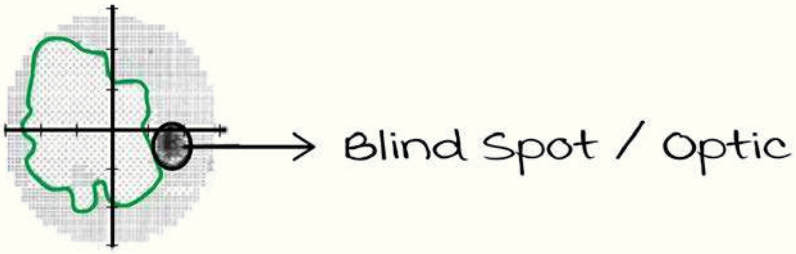
Date: 17-12-2014
Time: 08:08
Age: 29

GHT Borderline *

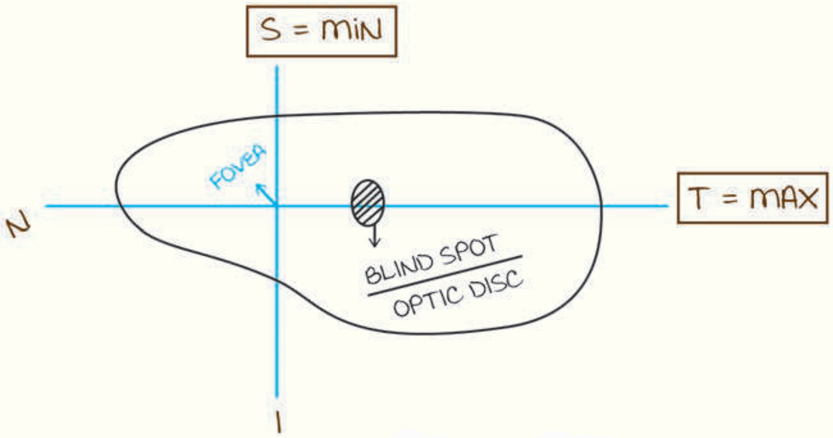
VFI 98%

MD -1.56 dB P < 10%
PSD 1.94 dB

→ Blind spot / optic disc



- Normal shape of the visual field :
  Horizontally oval with an Inferonasal notcht

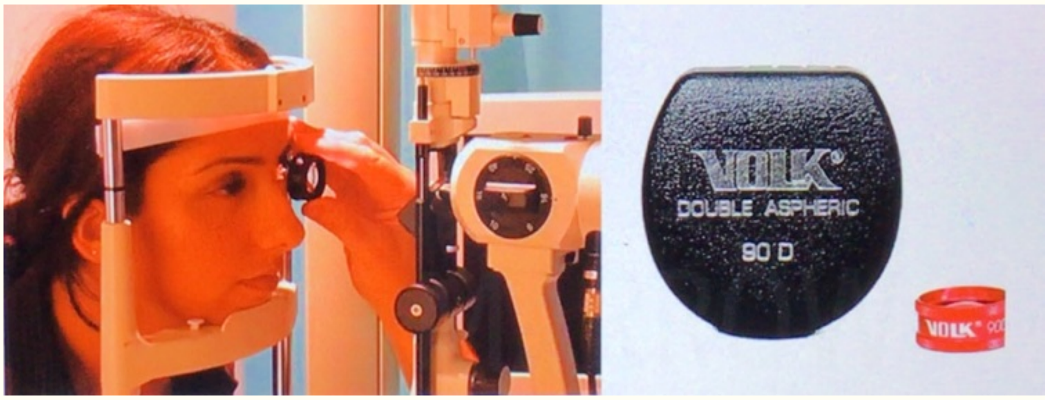


Optic Disc Examination

01:05:45

a. Ophthalmoscopy : (Direct)

b. Slit Lamp examination: uses a +90 Dioptre convex lens. *

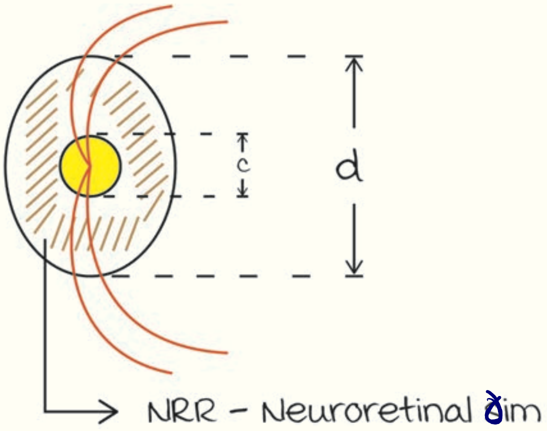


c. Optical Coherence Tomography(OCT)

d. Scanning Laser Polarimetery(SLP) : used to measure retinal nerve fibre layer (RNFL thickness)

e. Scanning Laser Tomography (SLO)

☆ Normal anatomy of Optic Disc



C = optic cup (central cavity/depression which allows vessels to travel through optic disc)

D = optic disc

- Normal C/D ration is 0.3 *
- Vertically oval shape
- Diameter of Optic Disc is 1.5mm *

Active space

Examination of Angle of Anterior Chamber

01:13:50

Oblique flashlight test:

Light is thrown is from the temporal side

↓

Temporal iris gets illuminated

Nasal iris also gets illuminated

↓

Anterior Chamber is deep

= Open Angle

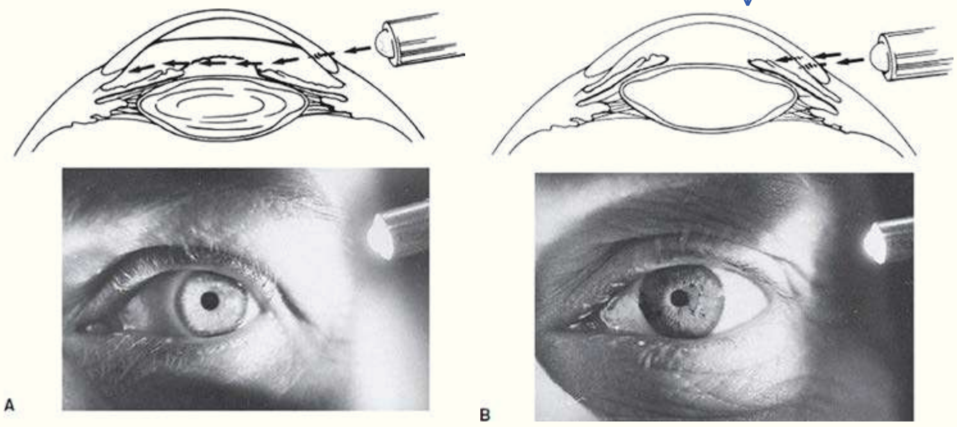
Shadow over nasal iris : Eclipse sign *

↓

Anterior Chamber is shallow

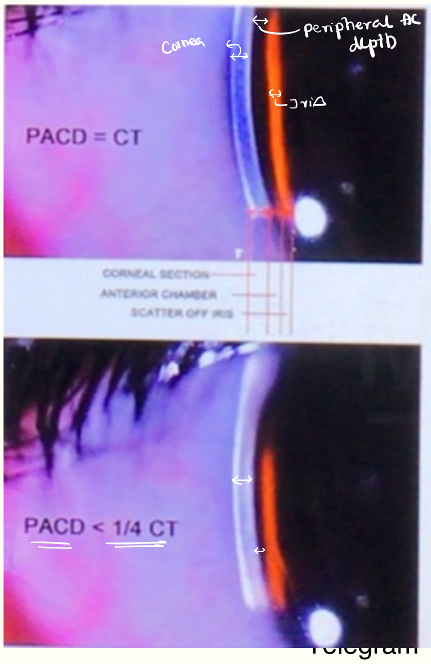
= Angle Closure ✓

↓



b. Van-Harick's method:

- Done on a slit lamp using oblique illumination
- Peripheral Anterior Chamber(PACD) depth is compared to Corneal Thickness(CT)



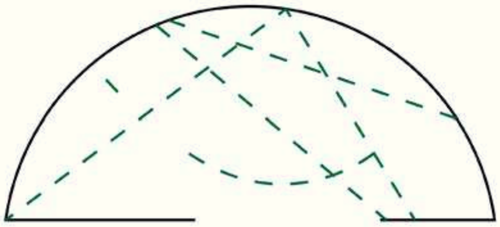
Open Angle

Angle Closure

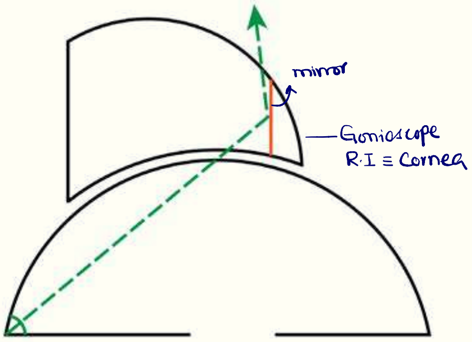
PACD/CT	Grade	remarks
≥1	4	Wide open angle
≤1/2 →1/4	3	Open angle
1/4	2	Should undergo Gonioscopy (to confirm)
<1/4	1	Very narrow angle (potentially occludable)

c. Gonioscopy: best

- Principle- elimination of Total Internal Reflection(TIR)



Light rays from angle of Anterior Chamber fall on cornea at an angle greater than 46 degree: Critical Angle and thus TIR keeps on occuring .



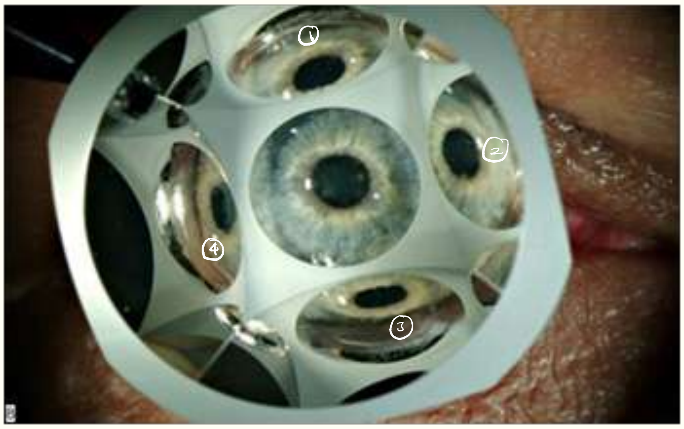
Types

Direct (Goniolens)
e.g. Koeppe used
in infants

Indirect (Gonioprism)
e.g. Zeiss four mirror,
Susman, Posner

Active space

- Zeiss four mirror :



- During Gonioscopy, trying to visualize the structure of the angle of Anterior Chamber:

↓

"Posterior to anterior"

Can't : Ciliary body band (CBB)
See : Scleral spur (SS)
This : Trabecular meshwork (TM)
Stuff : Schwalbe's line(peripheral termination of Descemet's membrane) – Cornea

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

Grading

- Scheie – b/o No of structures visible

All 4 → Gr I

Ciliary body band → Gr II
– obscured

CBB + SS + Some T.M – Gr III
– obscured

Only Schwalbe's line – Gr IV
visible

Shaffer: b/o degree of Angle

$35-45^0$ – Gr 4 → open

$25^0 - 35^0$ – Gr 3

$10^0 - 25^0$ – Gr 2

$<10^0$ – Gr 1 → closed

Spaeth: 9 parameters

Active space

Telegram--@latestpgnotes

GLAUCOMA: POAG AND PACG

Primary open angle glaucoma - POAG 00:00:07

It was also known as chronic simple glaucoma(not used any longer).

MC in old age > 50 years.

It is bilateral and symmetrical.

Family history is present - genes associated are :

- optineurin ★ AIIMS 14
- myoc
- WDR 36

Symptoms

- Headache
- Delayed dark adaptation
- ⇒ Frequent changes of presbyopic glasses

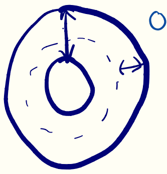
Signs of PAOG 00:06:31

IOP (intraocular pressure) -

> 21 mmHg

⇒ > 5 mmHg difference between both eyes (even if IOP is within normal limits)

⇒ > 8 mmHg difference in diurnal variation



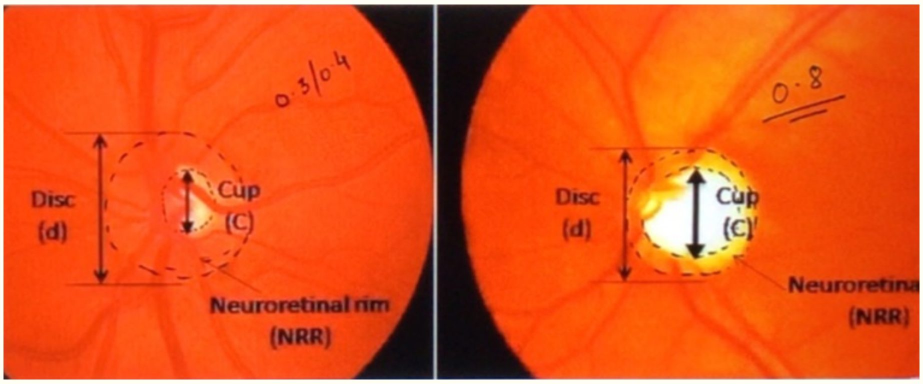
Optic disc signs :

↳ Contain N/v fibres ⇒ N/v fibres are damaged.

- Loss of neuroretinal rim(NRR) - initially, it affects inferotemporal NRR called polar notching and lastly, nasal NRR is affected.
- Ventral cupping

The nerve fibres are damaged and are lost in the neuroretinal rim which causes the enlargement of the cup called cupping, c/d ratio > 0.5.

Active space



Normal

ventral cupping

Other Signs of PAOG

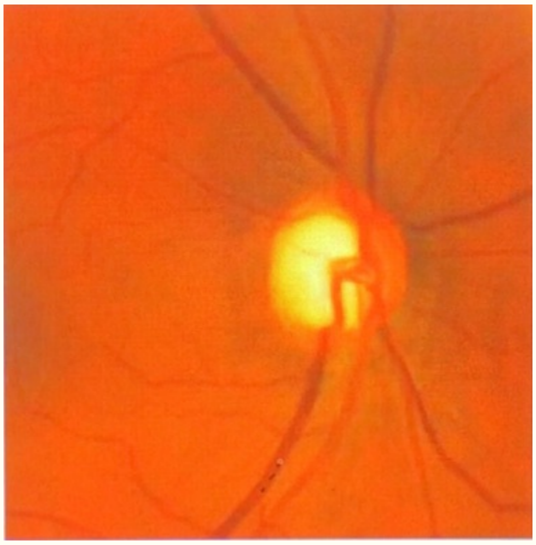
00:11:40

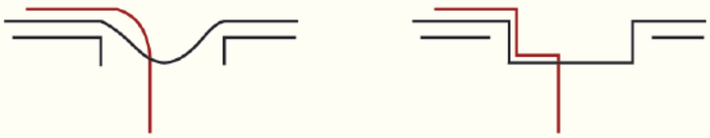
→ lamina Cribrosa

Laminar dot sign - Fenestration of sclera are visible as grey white dots.

Splinter haemorrhages on optic disc surface.

Bayonetting sign - double angulation of blood vessel.





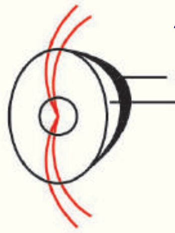
Normal

Bayonetting Sign

RNFL :

Retinal nerve fibre layer: wedge shaped defects - This is for early detection of glaucoma

Peripapillary atrophy



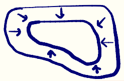
2 zones

α zone - Specific for Glaucoma

β zone - alse seen in myopia

Visual field changes

00:18:04



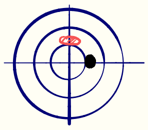
Isotopercontraction - earliest VF defect

↓

Baring of blind spot

↓

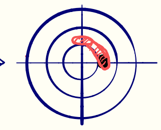
Paracentral scotoma



occur in Bjerrum's area

↓

Seidel's scotoma (sickle shaped) →

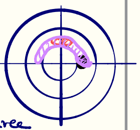


↓

Arcuate / Bjerrum scotoma

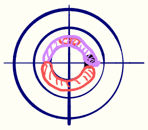
b/c it involves arcuate fibres

↳ b/c they are located in Bjerrum area



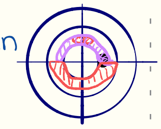
↓

Double arcuate / ring scotoma



↓

Step defects - due to unequal contraction /loss of nerve fibres in upper & lower parts It can be central or roenne's peripheral

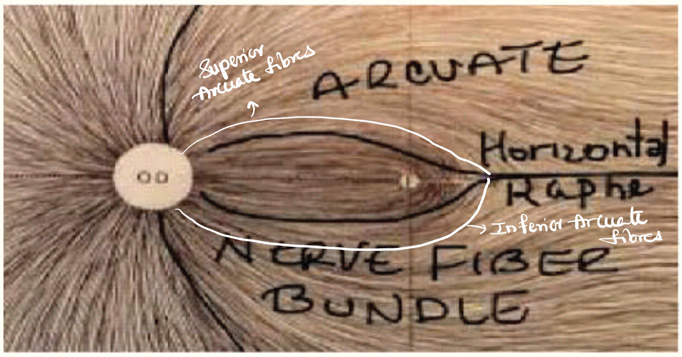


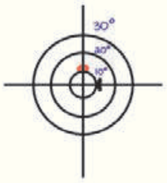
↓

Loss of central visual field

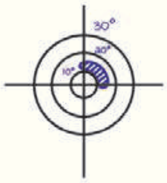
↓

Loss of temporal vision (last affected)

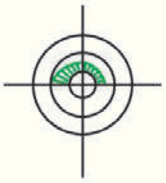


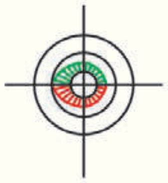


Paracentral Scotoma



Seidel's Scotoma


Arcuate Scotoma


Double arcuate/ ring Scotoma

Medical treatment of POAG -PGF2 analogues

00:29:09

Route of administration – Topical

1) PGF2 analogues : DOC

e.g., latanoprost, bimatoprost

mechanism of action – increased uveoscleral outflow

Dosing – OD at 9 P.m *

Ocular S/E – hyperpigmentation of iris

Hypertrichosis (growth of eyelashes)

Uveitis

→ Cystoid macular edema

Reactivation of herpetic keratitis*

Note: bimatoprost is FDA approved for treatment of hypo trichosis

Medicaltreatment of POAG - β blockers, α agonists

00:35:12

β blockers:

- Timolol – BD
- Betoxalol

MoA : ↓ aqueous secretion

Ocular S/E : 1) Blepharoconjunctivitis

2) NLD blockage

3) Corneal Anesthesia

4) Betaxolol → Aphakic CME

CI: A – Arrythmia

B – Bronchospasm

C – CHF

D – DM

Active space

α AGONISTS :

Apraclonidine

↓ Aq. secrez
↑ Trab outflow
MoA

Follicular conj'itis
Mydriasis
Eyelid Retraction
Ocular s/e

– DOC : for IOP elevation post laser Rx

Brimonidine

↓ Aq secrez

CI (ABCD)
· Drowsiness
· Depression
· Apnoea
· Bradycardia

CNS suppresant
⇓
CI : Children

Medical treatment of POAG -Carbonic anhydrase inhibitors& new drugs

00:43:06

Carbonic anhydrase inhibitors

E.g., acetazolamide (not give topically), brinzolamide, dorzolamide (given in children)

mechanism of action - decreased aqueous solution

→ Contraindicated in sulfa allergy

New drugs

1. Nitric oxide donating prostaglandin analogues

E.g., latanoprostenebunod - when administered, it is broken into

Latanoprost ✓
→ Increases uveoscleral outflow

butadeniol ✓
Donates NO
✱ Relaxes trabecular meshwork

- ↓NE Transport → ↓Aq prodn
- ⊕ Actin-myosin Contraction → ↑ Trabecular outflow

2. Rho Kinase inhibitors
E.g., netarsudil
mechanism of action - decreases aqueous production by inhibiting norepinephrine transport and increases trabecular outflow by promoting actin - myosin contraction.
Ocular S/E - cornea verticillata.

Surgical treatment of POAG

00:49:07

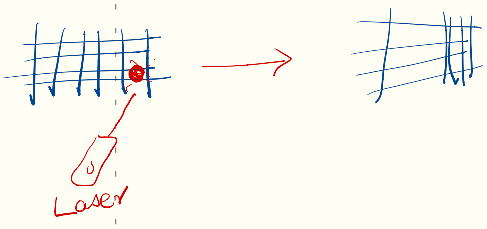
1) Laser trabeculoplasty(LT)

Two types

ALT
Argon LT
510 nm

SLT
selective LT
Selective laser is applied to pigmented trabecular meshwork

Photocoagula



Frequency doubled
ND:YAG laser (532 nm)
$f \left(\propto 1/\lambda \right) \Rightarrow 2f \rightarrow \lambda/2 = \frac{1064}{2}$

mechanism of action of argon and ND: YAG laser is photocoagulation
SLT is the treatment of choice for prophylaxis in the other eye.
(as POAG = B/L)

2) Trabeculectomy

A fistula is created between anterior chamber and sub tenon's space.
⇒ mcc of failure - fibrosis of fistula - to prevent fibrosis, use anti-metabolites (mitomycin, 5FU)

Non penetrating surgeries : (T.M = not penetrated - is bypassed)
- Deep sclerectomy ✓
- Viscocanalostomy ✓

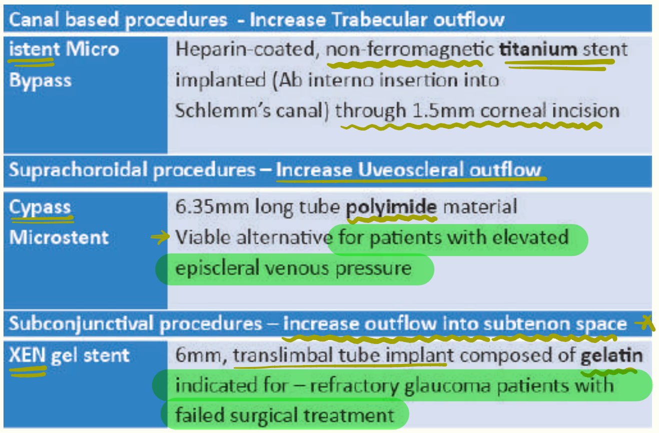
They have less chances of postoperative complications.

Active space

Surgical treatment of POAG– MIGS &Glaucoma drainage devices

00:56:23

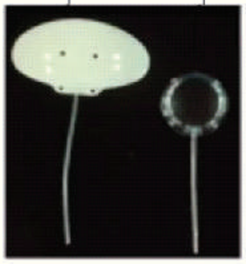
micro incision glaucoma surgeries (MIGS)

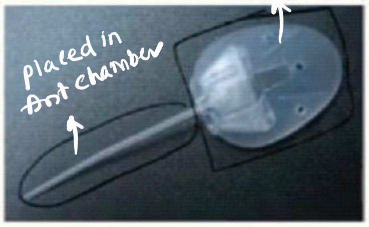


Canal based procedures - Increase Trabecular outflow	
istent Micro Bypass	Heparin-coated, non-ferromagnetic **titanium** stent implanted (Ab interno insertion into Schlemm's canal) through 1.5mm corneal incision
Suprachoroidal procedures – Increase Uveoscleral outflow	
Cypass Microstent	6.35mm long tube **polyimide** material Viable alternative for patients with elevated episcleral venous pressure
Subconjunctival procedures – Increase outflow into subtenon space	
XEN gel stent	6mm, translimbal tube implant composed of **gelatin** indicated for – refractory glaucoma patients with failed surgical treatment

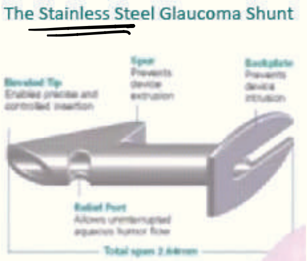
Glaucoma drainage devices/ seton surgery

It is done in refractory, un controlled glaucoma and glaucoma with aniridia





AHMED GLAUCOMA VALVE


The Stainless Steel Glaucoma Shunt
Beveled Tip
Enables precise and controlled insertion
Spur
Prevents device extrusion
Backplate
Prevents device intrusion
Radial Port
Allows uninterrupted aqueous humor flow
Total span 2.64mm

Express mini Glaucoma shunt

AIIMS -14

Primary angle closure glaucoma – PACG

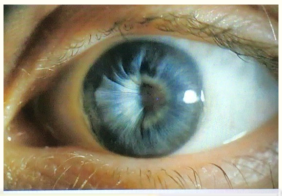
01:01:06

It is also known as acute congestive glaucoma

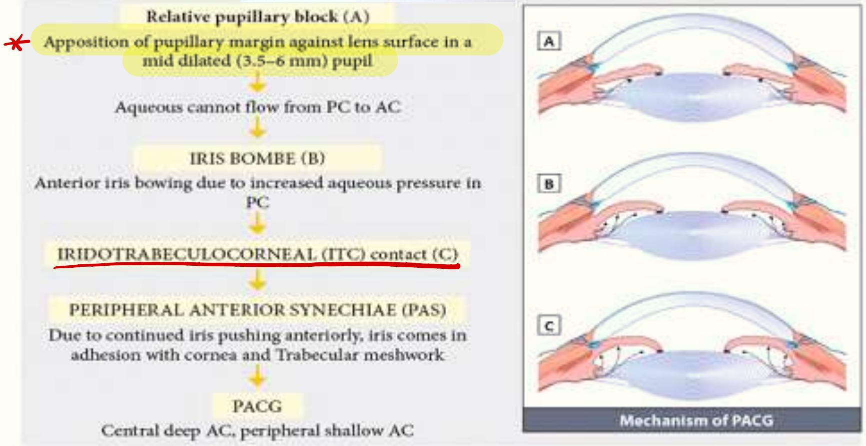
MC in young adults 20- 40 years

mechanism of PACG

Telegram--@latestpgnotes



Iris Bombe



Relative pupillary block (A)

* Apposition of pupillary margin against lens surface in a mid dilated (3.5–6 mm) pupil

↓

Aqueous cannot flow from PC to AC

↓

IRIS BOMBE (B)

Anterior iris bowing due to increased aqueous pressure in PC

↓

IRIDOTRABECULOCORNEAL (ITC) contact (C)

↓

PERIPHERAL ANTERIOR SYNECHIAE (PAS)

Due to continued iris pushing anteriorly, iris comes in adhesion with cornea and Trabecular meshwork

↓

PACG

Central deep AC, peripheral shallow AC

Mechanism of PACG

Risk factors & Classification of PACG

01:06:03

Risk factors

- Hypermetropia (small axial length)
- Small cornea (diameter decreased)
- Shallow anterior chamber
- Females
- * Plateau iris

All these factors can be precipitated by dim light ⇒ pt -advised not to visit cinema hall

Atropine is contraindicated in PACG

↳dilates pupil.

Classification of PACG

a) PACS primary angle closure suspect

Normal IOP

Normal optic disc

→ Apposition with > 3 quadrants – ITC cataract

b) PAC primary angle closure

→ Increased IOP

Normal optic disc

✓Peripheral anterior synechiae present

c) PACG

Optic disc and visual field changes are present. ✓

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Clinical features of PACG

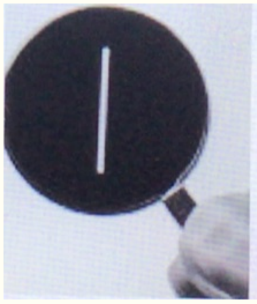
01:11:00

- Headache
- Loss/ blurring of vision
- Pain in the eye (severe)
- Nausea/vomiting
- Redness ✓
- Halos around light

The halos can also be seen in :

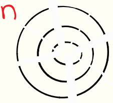
1. mucopurulent conjunctivitis(due to discharge) – on washing the eye, discharge is washed away and halos disappear.
2. Cataract
3. PACG

To differentiate between cataract and PACG – fincham's test using stenopaeic slit → to Dx Cataract



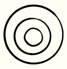
on rotation

If halos appear broken



Cataract

if halos appear intact



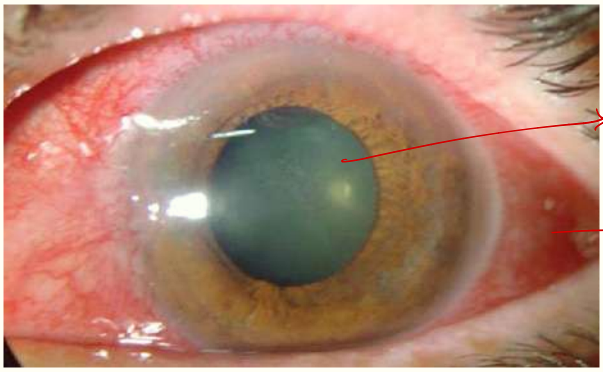
PACG

→ PACG is a diagnosis of exclusion

Signs OF PACG

01:16:03

1. Increased IOP >40 mmHg – causes severe pain in the eye ✓
2. On digital tonometry- rock hard eye*
3. On torchlight test- eclipse sign (due to shallow anterior chamber)
4. Pupil-vertically oval
   mid dilated
   Fixed – due to posterior synechiae



ACG
mid dilated pupil

Redness
↓ Uveitis → miosis
↘ Conjunctivitis → Ⓝ pupil

Slit lamp examination of PACG

01:19:53

Halos around light (prismatic effect) → Blurring of vision

→ 1. Edematous cornea/hazy cornea (IOP >40 → Tight Jns of Endothelium =lost)
2. Iris atrophy (d/t pressure)
3. Posterior synechiae-adhesion between pupillary margin and ens
4. Cataract known as glaucomflecken
   Anterior capsular opacities due to lens epithelial ischemia and necrosis

Treatment of PACG

01:25:38

· Acute → Immediate Redn of IOP

DOC Acute ACG : IV 25% Mannitol over 30min
or
Acetazolamide 250mg tab ② stat

↓

DOC ACG : Pilocarpine : miotic
– pulls iris away from angle

↓ IOP controlled

TOC : Laser Iridotomy
– usually b/w 11'o clock – 1'o clock
(Ⓡ : Upper part of Iris is Covered by eyelid (upper)
i.e diplopia = prevented)

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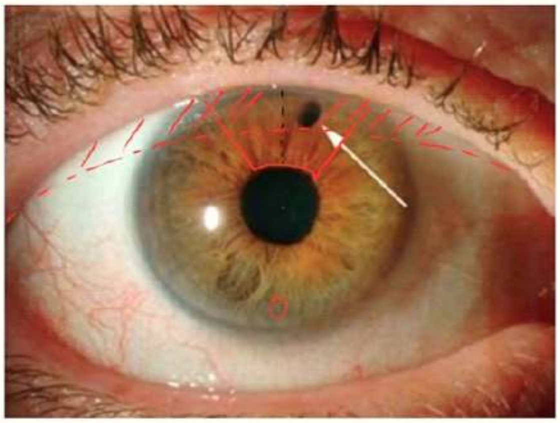

	OAG	ACG
	Chr.	Acute
Path:	TM-outflow.	Iris - Inflow
Rx:	@TM	@Iris

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GLAUCOMA: SECONDARY GLAUCOMA, CONGENITAL GLAUCOMA

Secondary glaucoma

00:00:06

Lens induced Glaucoma :

a. Phacolytic glaucoma

- Caused by morgagnian cataract
- Secondary open angle glaucoma.

b. Phacomorphic glaucoma

- Caused by Intumescent cataract (The swollen lens touches the pupil).
- Secondary angle closure glaucoma.

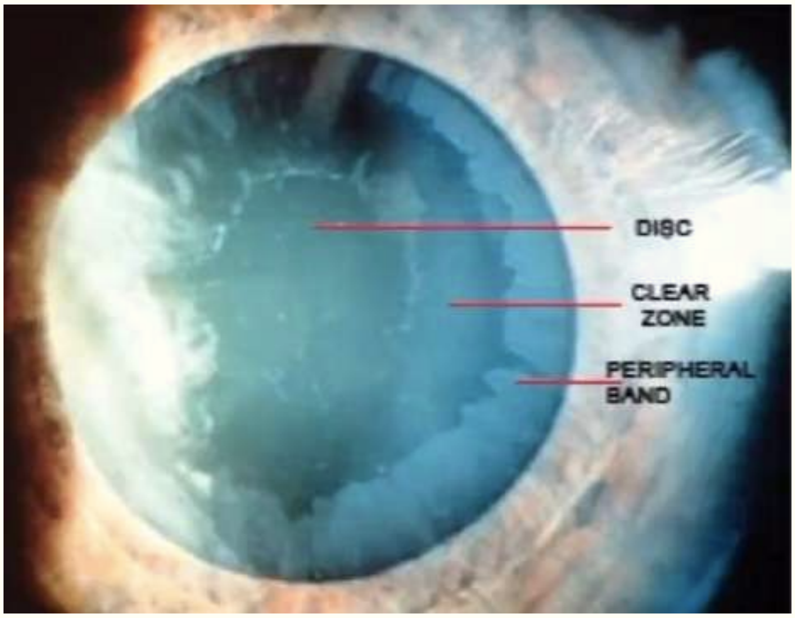
c. Phacotopic glaucoma

- Occurs in case of microspherophakia : will marchesani sx
- Secondary angle closure glaucoma.

Pseudoexfoliation glaucoma :

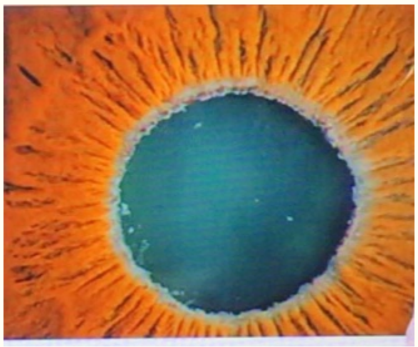
- Secondary open angle glaucoma.
- Occurs due to LOXL1 gene mutation. *
- Clinical features :

  a. Target sign : On lens



Active space

b. Fnocks : Whitish deposits on pupillary margin.



c. Poor mydriasis

d. Sampaolesi line : Deposition anterior to Schwalbe's line.

Pigmentary glaucoma :

- ✓ Secondary open angle glaucoma.
- Most common in myopes who are young males.

Clinical features	Deposition of pigment on :
a. Krukenberg spindle	Corneal endothelium
b. Zentamyer line	Posterior surface of lens near insertion of zonules
c. Sampaolesi line	

Secondary glaucoma: Steroid induced, Malignant

00:09:05

Steroid induced glaucoma :

- Due to increased production and decreased destruction of extracellular matrix of Trabecular meshwork.
- Leads to Secondary open angle glaucoma. ✓
- Steroid responders :

Active space

After steroid infused

High	Moderate	Non
>31 mm Hg IOP	25-31 mm Hg IOP	<20 mm Hg IOP
or	or	or
>15 mm Hg increase in IOP	6-15 mm Hg increase in IOP	<6 mm Hg increase in IOP
Cant prescribe steroids		↓ Safe

- Treatment : Beta blockers or Laser trabeculopathy

Malignant glaucoma :

- Also known as Ciliary block glaucoma / Aqueous misdirection syndrome / Inverse glaucoma.
- The cause is incisional or laser surgery.
- Mechanism : Anterior rotation of Ciliary processes leading to Cilio-lenticular block.

↓

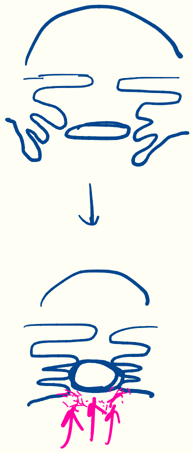
Posterior flow of the aqueous.

↓

Iris-lens diaphragm gets pushed forward.

↓

Secondary angle closure glaucoma (without pupillary block).



- Treatment : Atropine (Cycloplegic) (No use of pilocarpine)

Relaxation of ciliary muscles. : Ciliary lenticular block → Reversed / broken

↓

Stretching of zonules.

↓

Pulls the lens back

Childhood glaucoma

00:19:16

Types :	Onset	% of total cases
• Newborn	At birth	40
• Infantile	< 3 years	55
• Juvenile	3 – 16 years	5

Newborn and Infantileglaucomas are together known as * Primary congenital glaucoma. *

Primary congenital glaucoma :

- Onset <3 years of age.
- Mechanism : Trabeculo-dysgenesis (Open angle glaucoma).
- Symptoms – Triad of : Blepharospasm ✓
  * Photophobia (earliest) *
  Lacrimation

Active space

- Signs :

1. Earliest : Hazy cornea (~Blue) (child's eye = distensible) ↓ ↑IOP Endothelium break

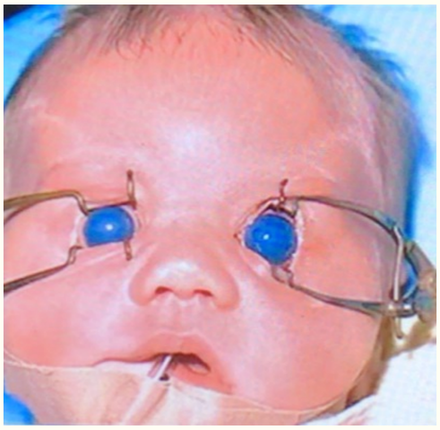
2. Haab's striae : Horizontal | Concentric breaks in descemet's membrane

3- Bupthalmos : * corneal d > 13mm (Ⓝ : 10-5mm)
  * Sclera - stretched thinned ↓ Blue sclera
  * AC : deeper
  * Myopia.

TOC : Trabeculectomy + Trabeculotomy

*If in Cong Glaucoma ↓ Pt : clear cornea
℞ : Goniotomy

Blue eyes – Congenital glaucoma



Gonioscopy
- Ant. Iris Insertion
- Barkan membrane (covering T.M)
- Loch-Ness monster phenomenon (loops of v/s)

Secondary congenital glaucoma

00:31:27

It is also known as Developmental glaucoma.

mechanism : Irido-corneo-trabeculo dysgenesis. (ICT) :

Causes :
- a. Sturge Weber syndrome
- b. Aniridia (75% cases causes glaucoma)
- c. Axenfeld Reiger syndrome
- d. Peters anomaly

Active space

OPTICS : TESTS FOR VISION AND OPTICS OF EYE

Introduction to optics 00:00:06

If a patient comes to the OPD :

- The vision of the patient is recorded.
- If the vision is normal, the optical system is assessed.
- If the recorded version is abnormal, the eye is checked for refractory errors.
- The refractory error is corrected using darkroom procedures.

Visual acuity 00:01:33

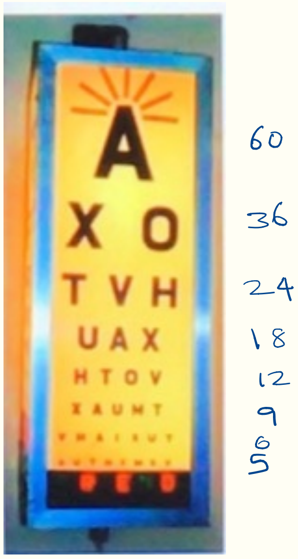
Components :

1. Minimum visible
2. Minimum resolvable :
   - Separation between two points such that they are the visible separately.
   - Minimum angle of resolution: 13 - 60 seconds of an arc. *
3. Minimum discriminable :
   - Offset 3-5 seconds of an arc known as Hyperacuity.
   - E. g : Vernier acuity ✓
     Stereo acuity ✓

Tests for vision : Snellen chart 00:04:50

Adults :

1. Snellenchart



- The topmost letter is the largest.
- Going down the letters become smaller.
- A standard Snellen chart has a fixed number written alongside each row of letters. The numbers denote the distance from which each specific letter can be read clearly by a normal person.
- Normal distance of testing : 6 metres/20 feet.
- If a patient's vision is 6/24 : A normal person can read the letter from a distance of 24 m, while the patient can read from a distance of 6 m.

  Denominator : Distance for normal person

  Numerator : Distance at which the patient reads

- method :

Patient stands at 6m and
reads letters from top to bottom

If the patient reads
all the row upto
7th row (6m)

6/6 : Normal vision

if the patient reads
only till the 4th row
(18 m)

6/18 : Decreased vision

Order of decreasing vision from the normal :

6/6 > 6/9 > 6/12 > 6/18 > 6/24 > 6/36 > 6/60 > 5/60 > 4/60 > 3/60 > 2/60 > 1/60 > Finger counting close to face (FCCF) > Hand movement perception

>Perception of light.

⇒ • Least vision recordable on Snellen chart : 1/60

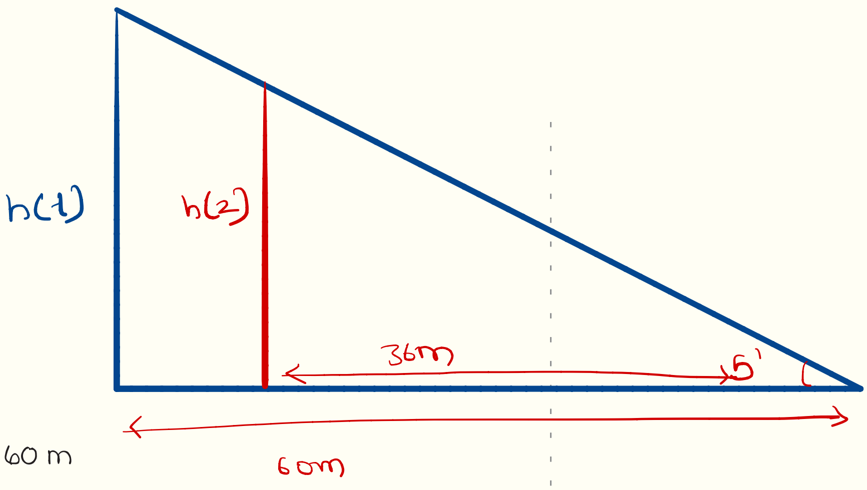
Principle of Snellen chart 00:17:18

* 5' of arc

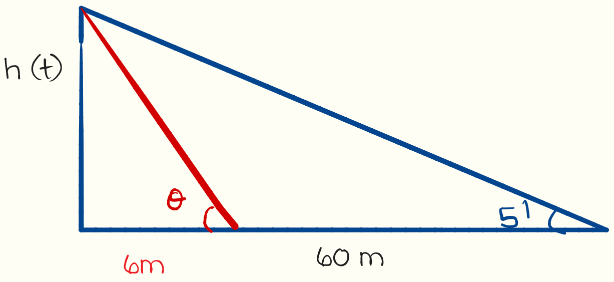
Each letter (optotype) subtends an angle of 5 minutes of arc at the nodal point of eye, when viewed from its respective distance.

Height of Topmost letter : h (t)

Height of 2nd row of letter : h(2)



The angle subtended by the topmost letter when viewed from 6 m :

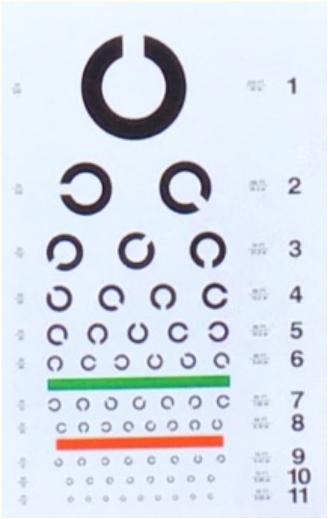


- From the diagram, it is evident that the angle increases.
- Angle subtended = $\frac{\text{Distance of respective letter} \times 5}{\text{Distance given for patient}}$; $\frac{60 \times 5}{6} = 50'$ of arc

Other tests for vision in adults

00:24:45

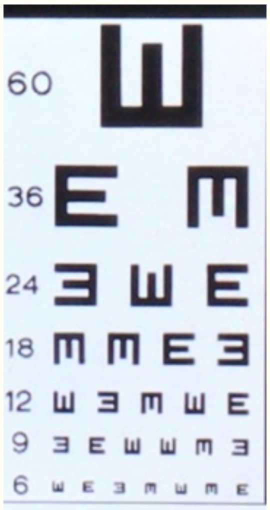
2. Landolt's C-ring test :



Active space

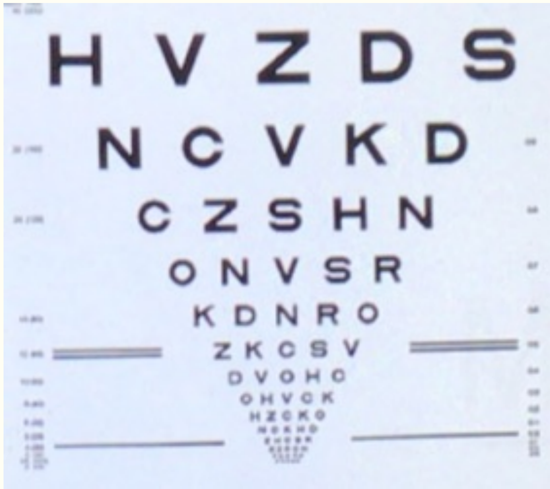
- Direction identification test.
- Used in illiterates.

3. Tumbling E chart :



- Direction identification.
- Used in illiterates.

4. Bailey - Love chart :



- Each row contains 5 letters.
- Designed on logarithm basis.

Tests for vision in children

00:28:30

Pre-school children 2-3 years old :

1. Broken wheel test :

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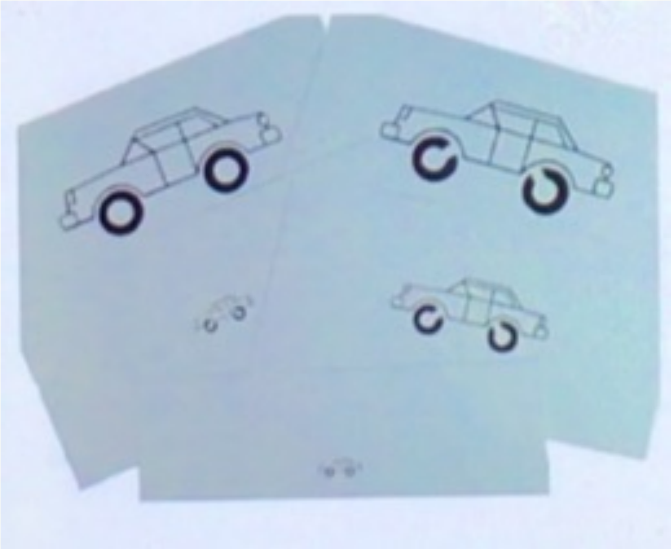
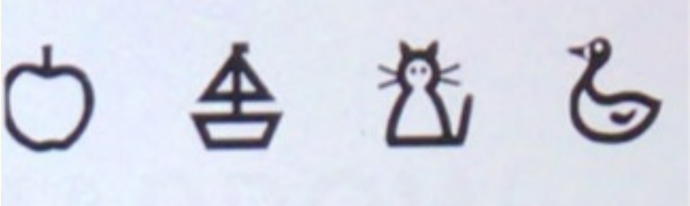


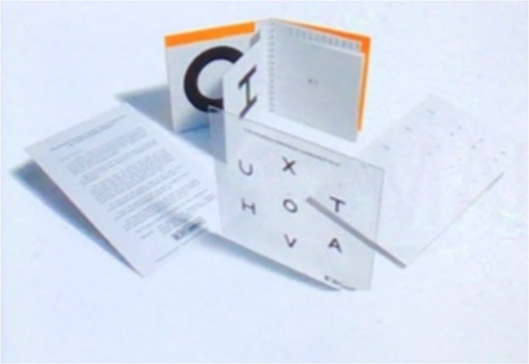
2. Kay picture test :



3. Sjogren's hand figure test
4. Allen pre-school test
5. Light house test

Children (2-5 years old, verbal) :

1. Sheridan-Gardiner HOTV test :

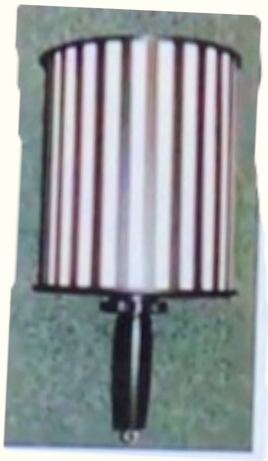


- The child is asked to match the letters from the book to the plate.

2. Keeler LogMAR Crowded test
3. STYCAR (Screening test for young children and retards)

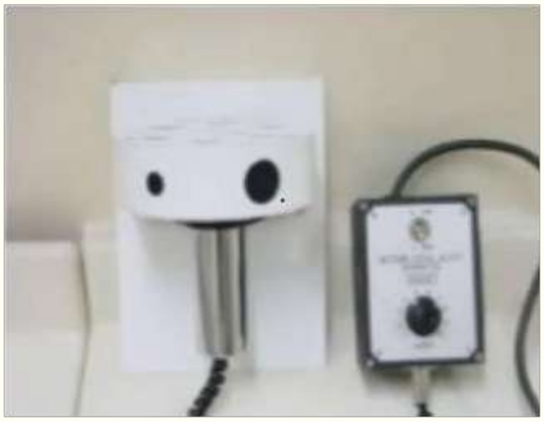
Infants :

1. OKN drum test :



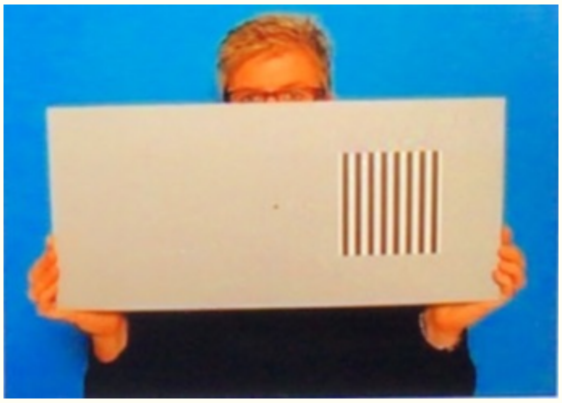
- Works by eliciting nystagmus.
- Resolution acuity method.

2. Catford drum test :



- Works by eliciting nystagmus.
- Detection acuity method.

3. Teller acuity cards :



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- An infant prefers to fixate on a striped pattern versus a homogenous (plane) surface.

4. Keeler gratings :

- The principle is same as that in Teller acuity cards.
- Cards are printed with a circular patch with varying thickness of stripes.

5. Cardiff acuity test

- Vanishing optotype test.

6. VEP: visual Evoked potential

7. Indirect Assessment

a. Blink Reflex : Since birth

b- Menace Reflex : ⊕ @ 5m of Age

- Infant will close eyes when object approaches near eyes.

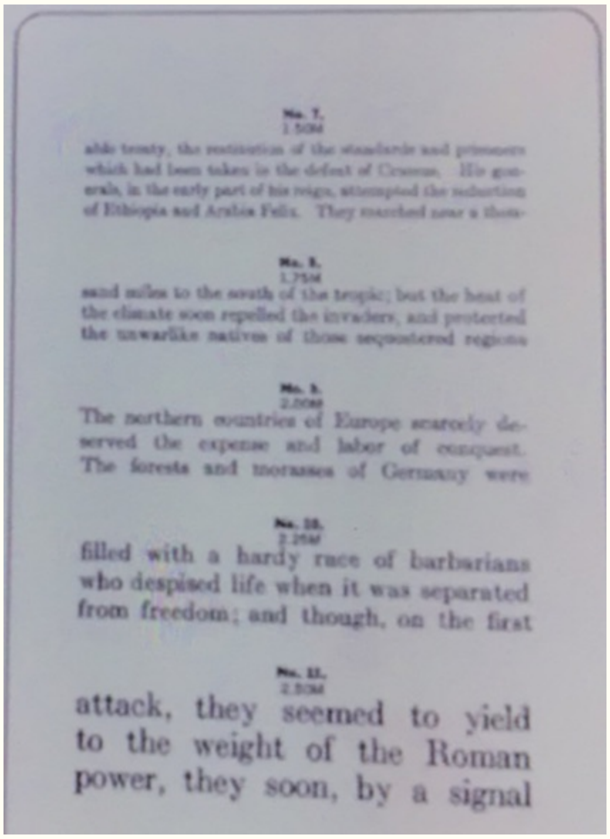
Active space

Tests for Near vision and Contrast sensitivity

00:38:08

Tests for near vision :

1. Jaeger's chart



No. 7.
1.50M

able treaty, the restitution of the standards and prisoners which had been taken in the defeat of Crassus. His generals, in the early part of his reign, attempted the reduction of Ethiopia and Arabia Felix. They marched near a thou-

No. 8.
1.75M

sand miles to the south of the tropic; but the heat of the climate soon repelled the invaders, and protected the unwarlike natives of those sequestered regions

No. 9.
2.00M

The northern countries of Europe scarcely deserved the expense and labor of conquest. The forests and morasses of Germany were

No. 10.
2.25M

filled with a hardy race of barbarians who despised life when it was separated from freedom; and though, on the first

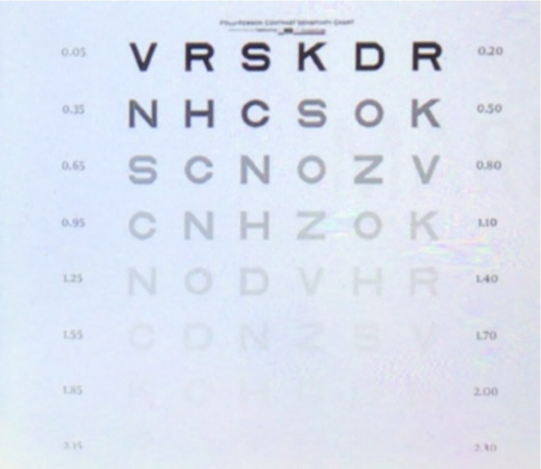
No. 11.
2.50M

attack, they seemed to yield to the weight of the Roman power, they soon, by a signal

2. Roman test
3. Snellen's near vision chart

Tests for Contrast sensitivity :

1. Pelli – Robson chart



2. Regan chart
3. Arden grating
4. FACT charts

Normal optical system of the eye

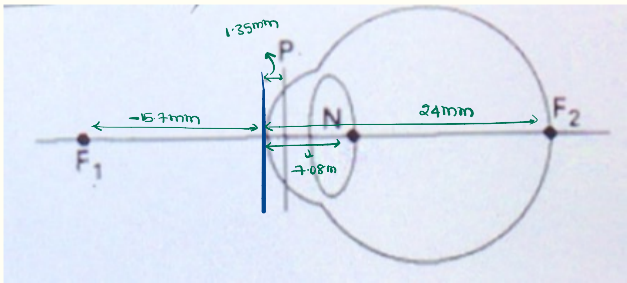
00:41:01

Optical system involves in converging the light rays and the refraction from 2 optical surfaces (Lens and Cornea).

Gullstrand's schematic eye : → Explains converging of rays

- Power of Cornea : + 43D to + 44D
- The cornea has 2 surfaces : Anterior surface : + 48.83D : 1 → 1.376 → convergence
  Posterior surface : – 5.88D (denser → Rarer medium) 1.376 → 1.336 ↓ divergence
- Power of Lens : + 16 to +19D

Listing's reduced eye : other factors deciding power of eye



All measurements
↓
from 1st Refracting surface
↓
Ant. surface of Cornea

P : Principal point
N : Nodal point
F1, F2 : Focal points

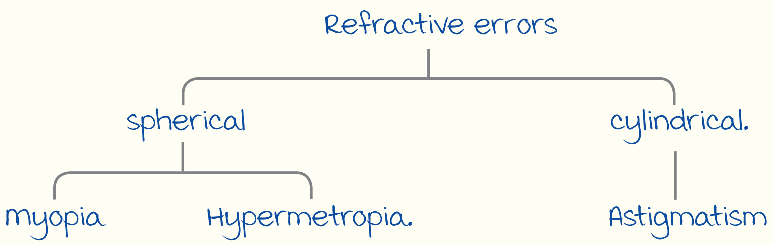
- The Principle point is located 1.35 mm posterior to the anterior surface of Cornea.
- The Nodal point is located 7.08 mm posterior to the anterior surface of Cornea.
- F2 is located 24 mm posterior to the anterior surface of Cornea.
- F1 is located 15.7 mm anterior to the anterior surface of Cornea.

- Refractory power depends on :
  1. Radii of curvature
  2. Difference in refractive index between various structures
  3. Axial length of eyeball

Active space

OPTICS: REFRACTORY ERRORS AND TESTS FOR REFRACTION

Introduction to Refractory errors 00:00:26



Myopia vs Hypermetropia: Mechanisms 00:03:32

myopia

Hypermetropia

	myopia	Hypermetropia
Power of the eye	more ☆ (ie wears ⊖ lens)	Less
Image formed	Infront of the Retina	Behind the Retina
mechanism /Causes	1. Axial myopia : • Axial length of eye : Increased • Imm increase in axial length = 3D myopia. • Buphthalmos 2. Curvatural myopia : • Curvature of cornea : Increased • Radius of curvature : Decreased • Keratoconus ✓ 3. Index myopia : • Refractive index of lens : Increased • Nuclear cataract	• Axial length of eye : Decreased • Curvature of cornea: Decreased • Radius of curvature: Increased • Refractive index of lens : Decreased

	4. Positional myopia : • Position of lens : Anterior • $P = 1/f$ f↓ ⇒ P↑ ✫ * • Weil-marchesani syndrome ↳ Ant/forward Ectopia lentis	• Position of lens : Posterior

Myopia vs Hypermetropia: Symptoms and Signs

00:16:06

	myopia	Hypermetropia
Symptoms	• Loss of far vision or short/Near sightedness • Floaters : d/t vitreous liquefaction	Loss of Near vision or Long/far sightedness • Asthenopia : tired eyes (Overworking Near Reflex)
Signs	* Eye ball : large ↓ ↓ scleral Rigidity * A.C : deeper * leads to Exophoria (far point : comes closer ↓ doesnot need to Accom for Near vision ↓ No convergence ↓ Convergence insufficiency ↓ Exophoria)	Eyeball : small shallower [R/F for ACG] Esophoria

Myopia vs Hypermetropia: Fundus signs

00:24:48

Fundus is a view of all the following structures visible in the posterior segment :

- Vitreous
- Retina

] Transparent

- Choroid : Red *

Fundus signs in Hypermetropia :

a. Pseudo-papillitis (Swollen disc)

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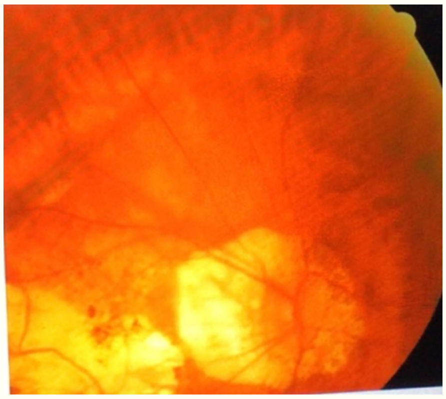
b. Shot silk fundus * – Shining

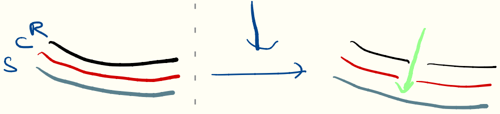
Fundus signs in myopia :

- They are seen only in cases of Pathological / Degenerative myopia : If myopia > 6D or axial length > 26 mm.

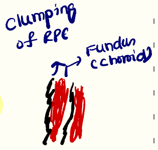
a. Temporal myopic crescent : Zone of Chorioretinal atrophy around the optic disc on temporal side.



d/t elongation → Retina & Choroid : atrophy



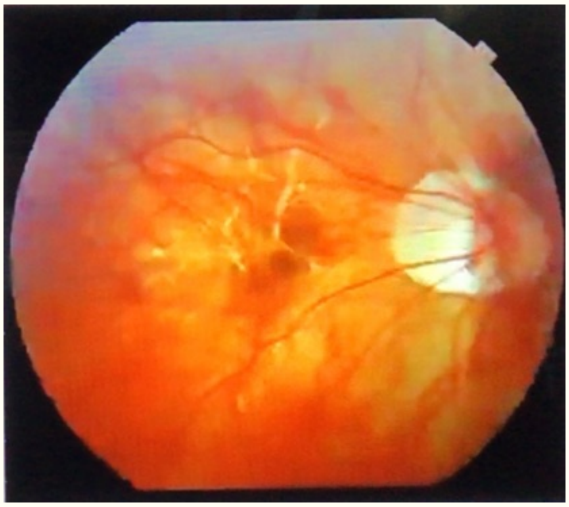
Sclera – visible (white)



b. Tesselated / Tigroid appearance of fundus : Occurs due to diffuse attenuation of retinal pigment epithelium.

c. Supertraction : Leads to tilted optic disc. *

d. Lacquer cracks : Fine breaks in the Bruch's membrane.



The above image shows :

1. Lacquer cracks
2. Temporal myopic crescent
3. Tilted optic disc

e. Foster fuch spots: Areas of subretinal haemorrhage at the macula. : dark Red spots

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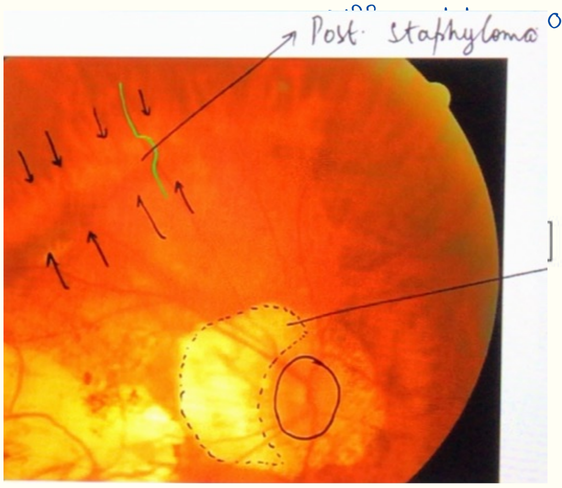
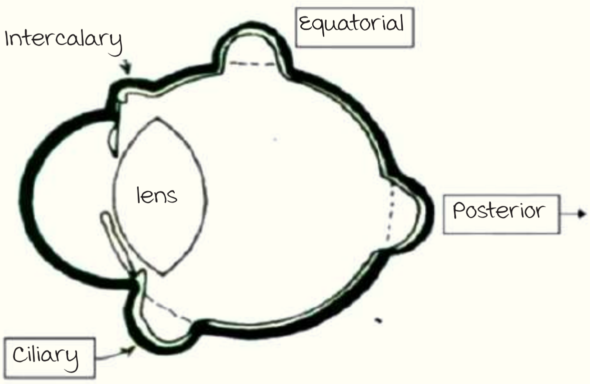
f. Posterior staphyloma: Bulging of sclera.

of Staphyloma :





- Intercalary : Bulging of limbus lined by root of iris.
- Ciliary : Bulging of sclera lined by ciliary body.
- Equatorial : Bulging of sclera lined by choroid.
- Anterior : Corneal bulging.

g. Vitreous hemorrhage ✓

⇒ h. Rhegmatogenous retinal detachment

Treatment of Myopia and Hypermetropia: Spectacles and Surgeries

00:45:10

Treatment of myopia : Spectacles

1. Spherical /Concave [–] or Diverging lens

Treatment of Hypermetropia : Spectacles

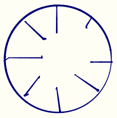
- Spherical /Convex [+] or Converging lens

2. Surgeries for Hypermetropia :

   a. Conductive Keratoplasty
   b. Thermal laser Keratoplasty
   c. Hyperopic LASIK

2. Surgeries for myopia :

   a. Radial Keratotomy : To flatten the curvature of the cornea.



   b. Laser surgeries :
      1. PRK : Photorefractive Keratectomy (-2D TO -6D)
      2. LASIK : Laser assisted in situ Keratomileusis : -2 → -13D

Excited dimer
↑
→ • Excimer laser : Argon Fluoride (193 nm)

- mechanism : Photoablate – flattening of cornea. ✓
  ↳ burn
- Steps : Use a microkeratone to create a nasally hinged epithelial flap

↓

Ablate of the corneal stroma
(Roughly 1D = 10μm ablation)

↓

Residual stromal thickness ≥ 250μm*

- Contraindications to LASIK :

→ – Age less than 18 years
– Spectacle power is not constant for past one year
– Corneal thickness < 450μ*

3. Latest surgeries :
   - Small incision Lenticule extraction (SMILE) or Refractiv Lenticule extraction (ReLEx)

Stroma

Lenticule

Active space

- On the removal of Lenticule, the thickness and curvature is decreased.
- → Laser used : Nd : Glass laser < SMILE FLACS
- Flapless and bladeless surgery

Treatment for Myopia

01:00:10

Other surgeries for myopia :

→ ↓ power by 16-18 D : i.e in High myopia

c. Fucala's operation: Extraction of clear lens and implantation of IOL.

d. Phakic refractive lenses (PRL)

- Implant an IOL – ICL (Implantable CollamerLens), over the normal crystalline lens. →high myopic

3. Contact lens: Preferred in High myopia

Astigmatism

01:04:32

Different refractory power in both principal meridian/ axis of eye.

Classification :

Regular
- When both principal meridian are 90° to each other

Irregular
- When both principal meridian are not at 90° to each other

With the Rule.

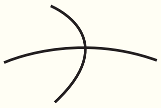
Vertical meridian is more curved.

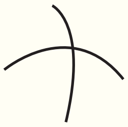
Against the Rule.

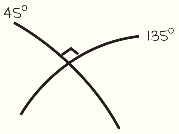
Horizontal meridian is more curved.

Oblique

Both Principal meridian are at 90° to each other but not vertical and horizontal.







Active space

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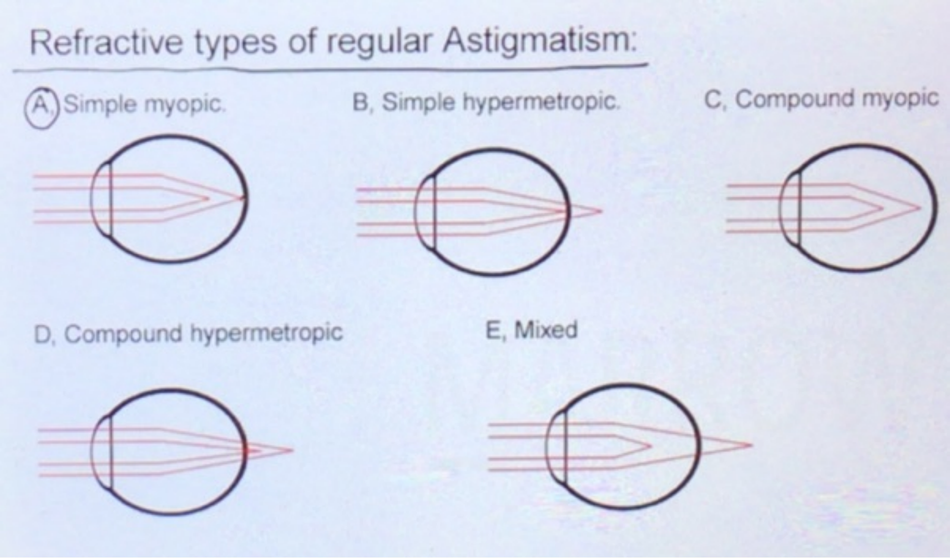


Refractive types of regular Astigmatism:

A, Simple myopic. B, Simple hypermetropic. C, Compound myopic

D, Compound hypermetropic E, Mixed

The most common type of Astigmatism: Compound myopic *

Astigmatism: Symptoms and Treatment

01:12:43

Symptoms of Astigmatism :

1. Asthenopia
2. Running together of letters : S → 8
3. High Astigmatism → tilting of head

Treatment of Astigmatism :

1. Spectacles : Cylindrical

Concave Convex b/o Refractive type

2. Sx :

a. Astigmatic LASIK

b. LRI - Limbal relaxing incisions

c. Ruiz procedure - post keratoplasty astigmatism

3. Toric contact lens

Active space

Reading a spectacle prescription

01:16:42

Examples :

- If the patient has been prescribed:
  - a. – 2D Spherical : myopia
  - b. + 0.75D Spherical : Hypermetropia
  - c. – 1.25D Cylindrical @ 90° : Simple myopic astigmatism against the rule.

    To Know if it is with rule or against the rule, mnemonic : WITH CV AGAINST CV.

	180°	90°
WITH	conCave [–]	convex [+]
AGAINST	convex [+]	conCave [–]

- d. + 2.50D Cylindrical @ 90° : Simple hypermetropic astigmatism with the rule.
- e. – 1D Spherical / – 1D Cylindrical @ 180° : Compound myopic astigmatismwith the rule.
- f. + 2D Spherical / + 1D Cylindrical @ 180° : Compound hypermetropic astigmatism against the rule.
- g. – 1D Spherical / + 2D Cylindrical @ 90° or + 1D Spherical / – 2D Cylindrical @ 90° : mixed astigmatism.

  Rules of mixed astigmatism :

  1. Cylinder and sphere of opposite signs.

  ★2. Cylindrical power>Spherical power.

h. – 3D Spherical / + 1D Cylindrical @ 90° :

- Not mixed astigmatism as Cylindrical power<Spherical power.
- Simple transposition is used :

  New sphere = Old spare + Old cylinder = –3 + 1 = –2D

  New cylinder = Opposite of old cylinder = –1D

  New axis = 90° to old axis = 180°

  After transposition, – 2D Spherical / –1D Cylindrical @ 180° :

  Compound myopic astigmatism with the rule.

Miscellaneous Refractory conditions 01:32:04

Aniseikonia :

- Distortion of vision.
- Different size and / or shape of image between both eyes.

Anisometropia :

- Different refractive power between both eyes.
- Significant only if the difference between both the eyes > 2.5D.
- E. g : If Right eye = +2D and Left eye = +6D.

  1D = 2% magnification.

  Therefore, Right eye : 4% magnification

  Left eye : 12% magnification

  ⇒ If the difference in image size is > 5%: diplopia occurs. ⇐

Emmetropia :

- Refractory power is normal.

Aphakia :

- Absence of lens.
- Causes Hypermetropia.
- Pupil : Jet black
- Purkinje images : Normally 4

  In Aphakia 2

I

III

IV

II

I : Anterior surface of cornea

II : Posterior surface of cornea

Active space

III : Anterior surface of lens

IV : Posterior surface of lens (concave- inverted image)

- Treatment :

⇒ 1. Treatment of choice : Implantation

2. Contact lens

3. Spectacles : Convex (+10D to +13D)

Side effects of a high powered spectacle :

a. Magnification - 25%

b. Pin-cushion defect (Spherical aberration)

peripheral - shrinks

Center : Clear

c. Jack in box phenomenon (Prismatic aberration) or Rowing ring scotoma.

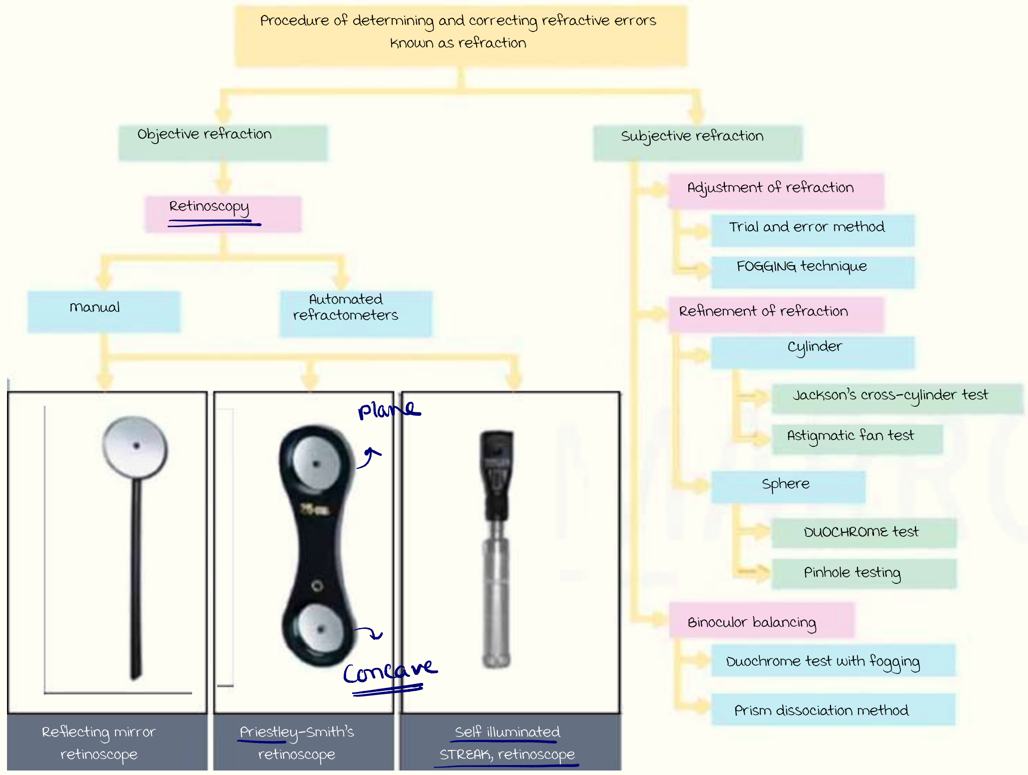
front side

2 refractory surfaces

Refraction and Retinoscopy

01:44:08

Refraction :



Reflecting mirror retinoscope

Priestley-Smith's retinoscope

Self illuminated STREAK retinoscope

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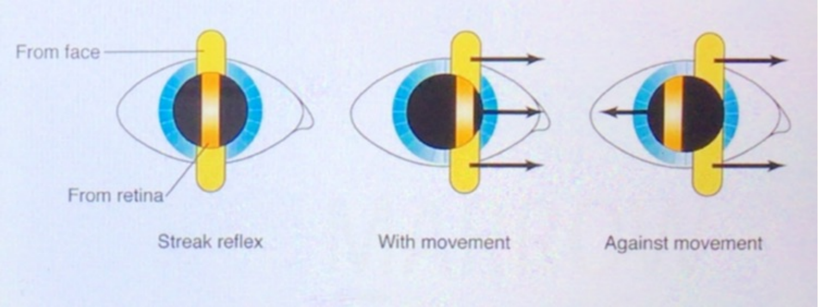
Retinoscopy :

Distance of examination : 1 metre.

Retinoscope used : Self illuminating streak Retinoscope.

method : movement of fundal red streak is noted in comparison to the movement of Retinoscope.

a. No movement : 1D myopia



b. With movement: < 1D myopia / Emmetropia ✓/ Hypermetropia ✓

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

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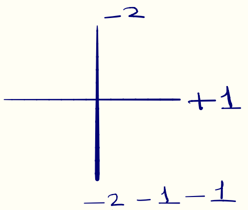
c. Against movement : > 1D myopia

Ex : ⇒ No movement : 1D myopia
Against 〃 : 2D myopia
↓
Neutralise c̄ −1D lens
⇓
Net : 1D ⇒ No movement
⇒ Hence : Confirmed : 2D myopic

Correction factors in Retinoscopy.

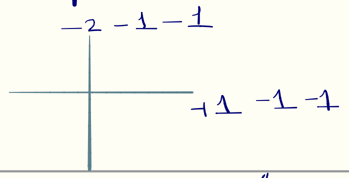
- Correction for distance = $\frac{1}{d(m)}$
- Correction for cycloplegia : Atropine : 1
  Cyclopentolate 0.75
  Homide − 0.5

(e) On Retinoscopy :



Distance : 1m
Using Atropine

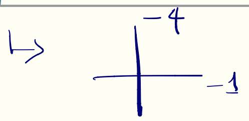
Corrected RF :



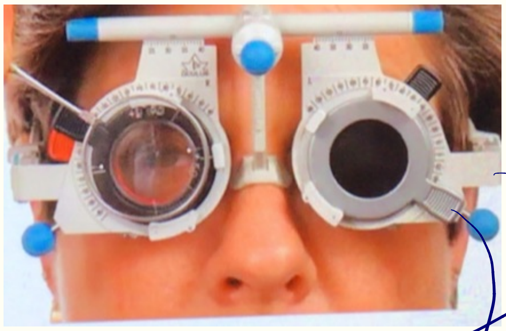
← CD = $\frac{1}{1}$ = 1
← CC = 1

Subjective Refraction

01:57:06



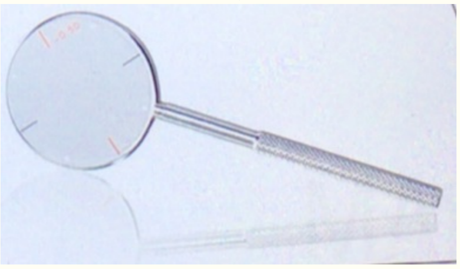
Trial and error method :



Trial frame
- Left eye is occluded.

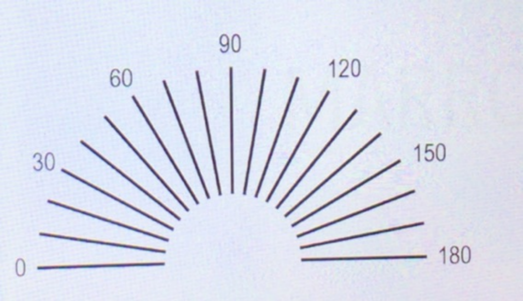
Refinement of Refraction :
1. Cylinder :

a. Jackson's cross-cylinder test :



- − 0.5D sphere with + 1.0D cylinder : Spherocylindrical lens.

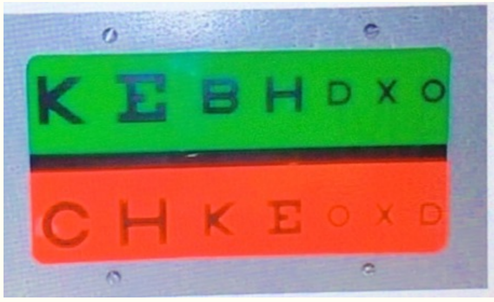
b. Astigmatic fan test :



- If all the lines are equal in intensity : No problem of axis
- If a particular is more clearly visible : Indicates the requirement of refinement of axis.

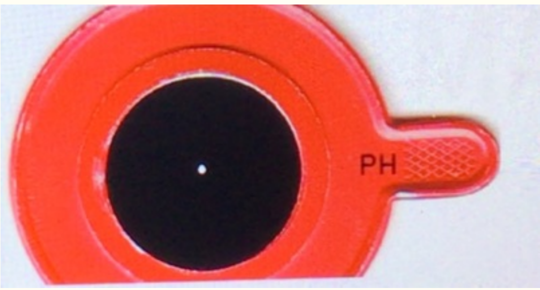
2. Sphere :

a. Duochrome test :



- If letters on green background are more clear : Residual hypermetropia.
- If letters on red background are more clear : Residual myopia.

b. Pinhole test :

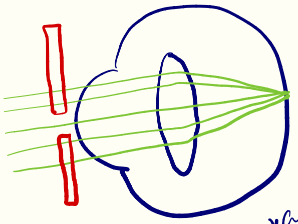


- Size of the pinhole : 1mm

Pinhole in front of the patient.

Vision improves. — * Residual refractory error (upto 4D) *

Vision decreases. — Mascular pathology.



Central light ray which is not refracted at all, is allowed to pass
↓
i.e Refractive error effect : Nullifies
↓
vision
- improves i/c/o R.E
- decreases i/c/o macular path.

- Use :
  1. ① Can refine any spherical error.
  2. (2) Diagnosis of refractory error up to 4D.
  3. (3) Diagnosis of macular disease.
  4. (4) Used as Low vision aids (LVA) : multiple pinholes
  5. (5) 2 point discrimination test : 2 pinholes

Binocular balancing : (i/c/o U/L Ⓝ on both eyes but binocular problem)

✓ 1. Duochrome test with fogging

✓ 2. Prism dissociation method

Emmetropization, Accommodation and Amaurosis 02:09:41

Emmetropization :

- New born has +2D to + 3D Hypermetropia (as eye is small).

↓

Achieves Emmetropia by 7 years of age. *

Accommodation :

- Maximum in children. ✓
- Least in old age.

Amaurosis :

- Complete loss of vision (unilateral / bilateral) without any findings / signs.
- Causes of bilateral Amaurosis :

1. Acute Nephritis
2. Uremia : Bilateral Amaurosis
   Pupil – Light reaction normal.

] Toxins act on higher visual centres.

Active space

Convex lens VS Concave lens

02:14:14

Convex	Concave
magnification	Diminished image.
If you look through a convex lens and move the lens ↓ Imagemoves in the opposite direction *	If you look through a concave lens and move the lens ↓ Image moves in the same direction
Uses : 1. Hypermetropia 2. Aphakia 3. Presbyopia → 4. Accommodative esotropia ℞ 5. Ophthalmoscopy	Uses : 1. myopia 2. Hruby Lens (-58.6D): ★ To visualise Fundus.

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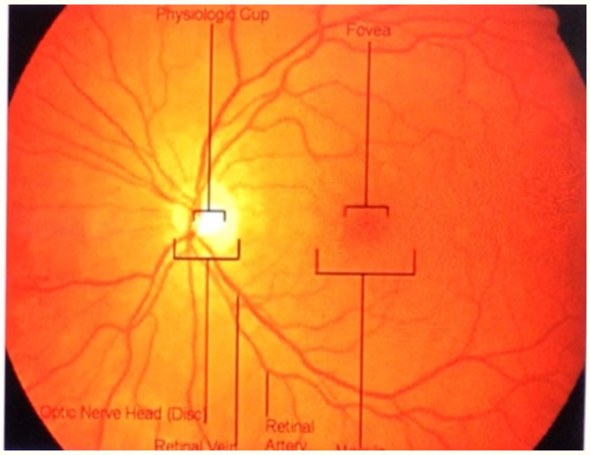
RETINA: ANATOMY AND INVESTIGATIONS

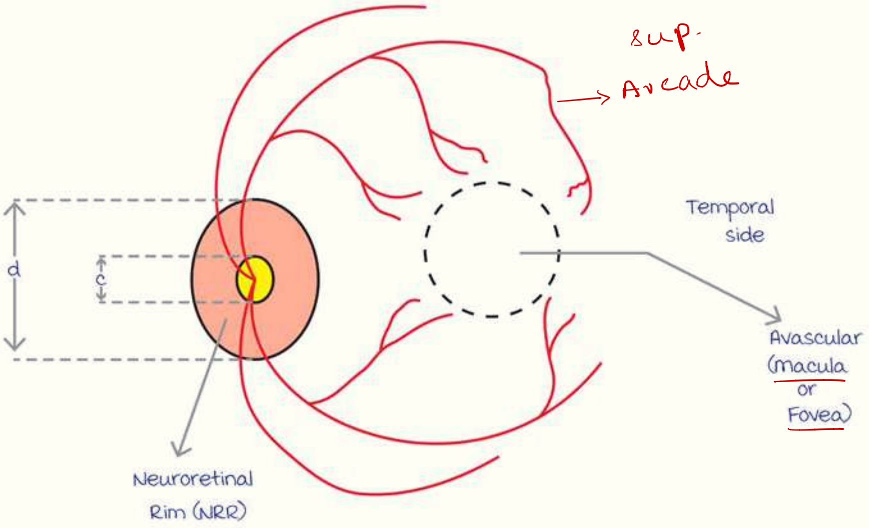
Introduction to Retina

00:01:27

Colour of the Retina : Transparent

Fundus : A view of Vitreous Transparent
Retina } Transparent
Choroid – Red





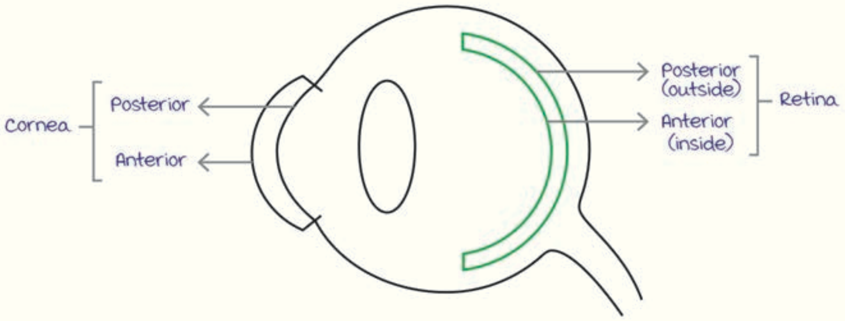
c/d ratio = 0.3

Optic disc diameter = 1.5mm

macula is temporal to Optic disc by 2 disc diameters.

Layers of Retina

00:09:25



mnemonic : ING I_2 O_2 Ep_2 or ING (IOP)2

Internal limiting membrane
- true B.M
- formed by footplates of muller cells

Nerve Fibre Layer

Ganglion cell layer – Ganglion cells – 3rd order Neurons

Inner Plexiform layer — Synapse : B : G

↳ synapse

Inner Nuclear layer : Bipolar cells – 2nd order Neurons

Outer plexiform layer — Synapse : Bipolar cells photoreceptors

Outer Nuclear layer

External limiting membrane (False BM)

Active space

Photoreceptors :

- Rods and Cones
- Ist order neurons

RetinalPigment epithelium (RPE)

- Forms the outer blood retinal barrier *
- Phagocytic function

Anatomy of Retina

00:22:41

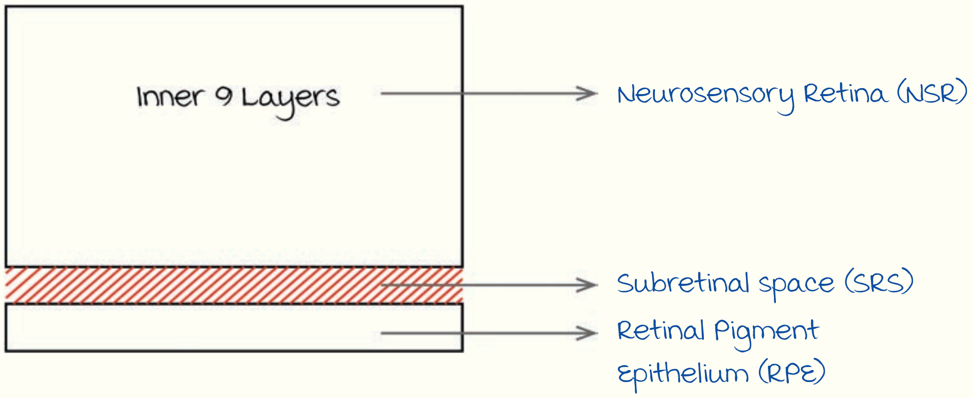
Ganglion cells are the sites of generation of Action potential. *

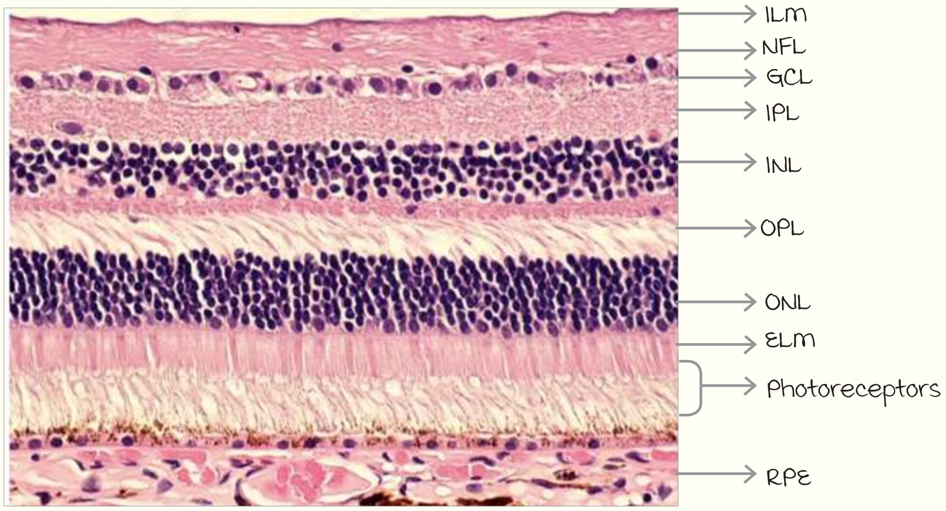
Photoreceptors generate local graded potential which is transmitted via electronic conduction.

Neuroglia cells in retina :

- Astrocytes
- Muller's cells
- microglia(redo

* All N/v fibres
↓
Optic N/v
↓
- 3rd order Neuron
- Axons of Ganglion cells





Blood supply of the Retina

00:30:25

- Inner 6 layers
  - Central retinal Artery
- outer 4 layers
  - short posterior ciliary artery

Inner blood retinal barrier: Formed by endothelial cells of retinal capillary in OPL. (Outer plexiform)

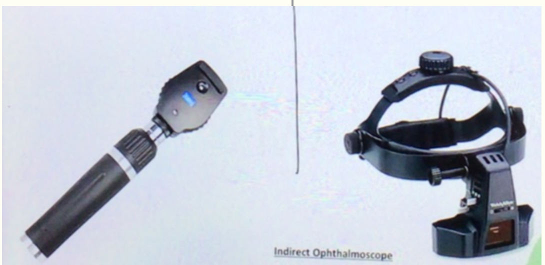
macula :

- Supplied by Central retinal artery.
- In some cases also by Cilioretinal artery (Branch of Posterior ciliary artery)*

Investigations : Ophthalmoscopy

00:33:58

1. Ophthalmoscopy
   - Fundus examination

Ophthalmoscopy :	Direct	Indirect
Features :	 Indirect Ophthalmoscope Looks like a Retinoscope (lens changed)	
magnification :	15 times (as OD : 1·5mm)	3 – 5 times
Lens :	Convex	Convex m/c power : +20D

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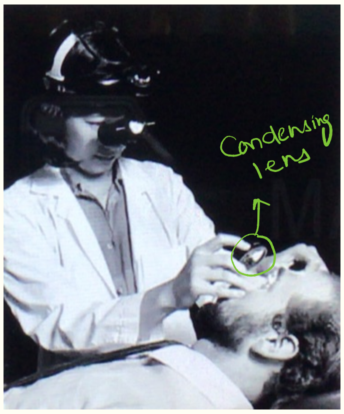
		- most common power: +20D  - Condensing lens in hand: +20D
Image formed :	Virtual and erect	Real and inverted (indirect)
Area visualised : magnification $\propto \frac{1}{\text{Area}}$	Smaller a. Central fundus or b. Optic disc and macula or c. 2 Disc diameters	Larger a. Central + Peripheral fundus or b. Optic disc ✓ + macula ✓ + Ora serrata ✓ or c. 8 Disc diameters
Stereopsis : Binocular depth perception.	Absent (monoocular)	Present
Distance of examination :	As close to the face as possible	An arm's length
Patient's position :	Sitting or Standing	Supine ☆
	Cannot be done if hazy media.	Can be done if hazy media.

Direct ophthalmoscopy : Glaucoma, Papillary edema → OD

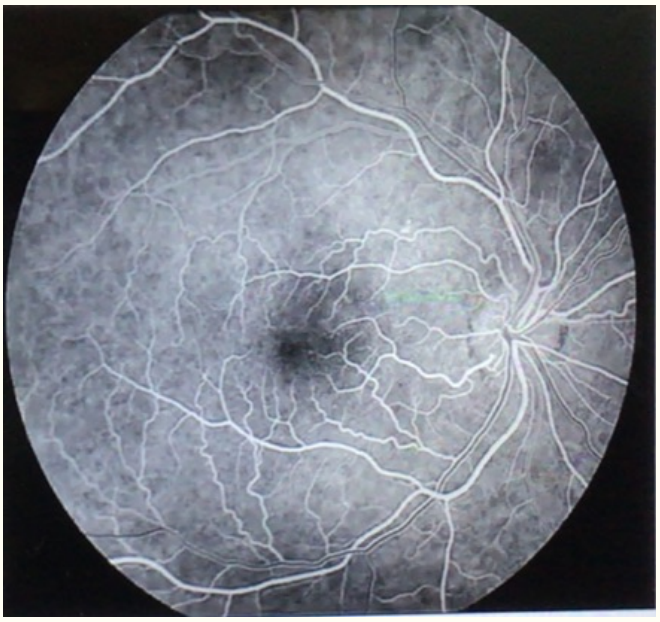
Indirect ophthalmoscopy : Retinal detachment

Investigations: FFA, ICGAand OCT

00:47:16

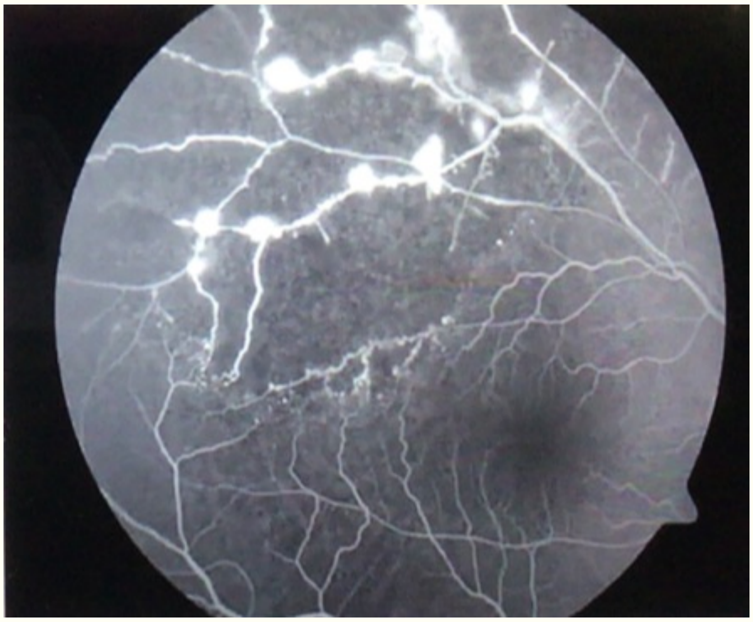
2. Fundus Fluorescein Angiography (FFA)

- Orange coloured dye
- Injected as 5% of 10 ml solution in Antecubital vein. *
- Blue light is shown: Dyefluoresce at 520 nm. *



Normal FFA

- Used to diagnose :
  a. Vascular pathologies



Leakage of dye : Hyperfluorescent areas.

Active space

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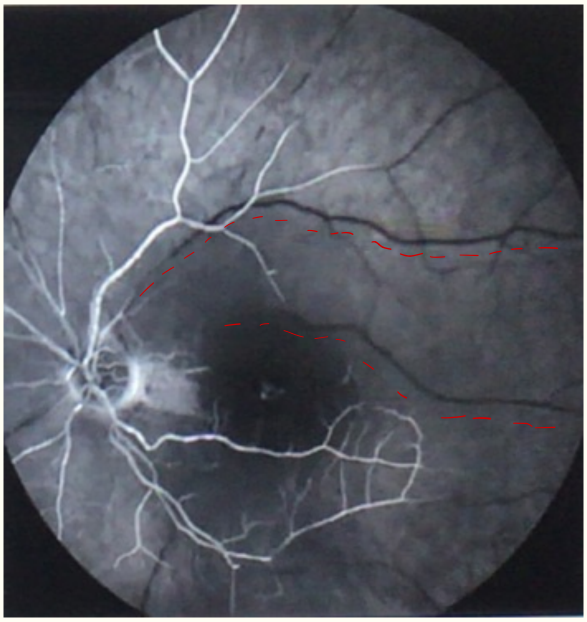
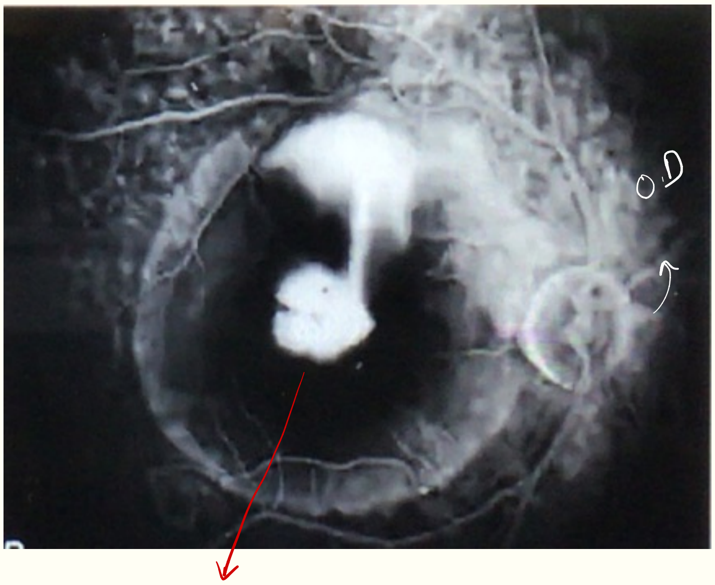


Hypofluorescent due to blockage.

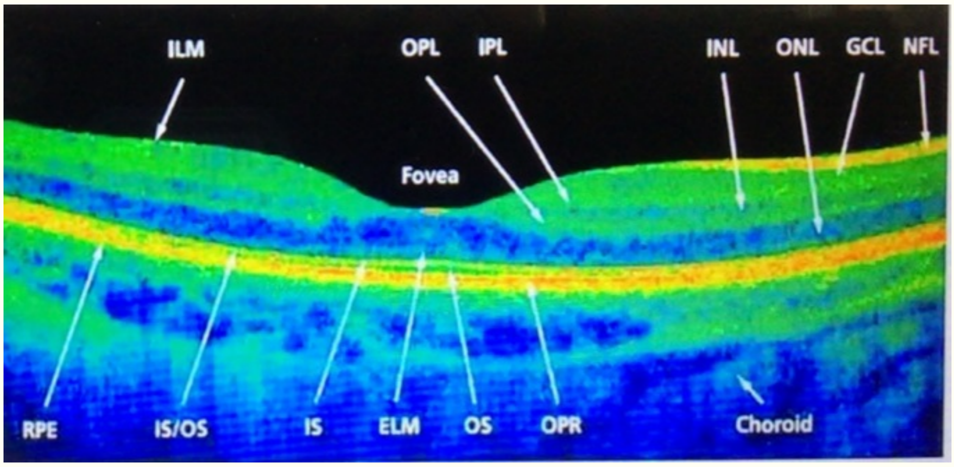
b. macular pathologies (Normal macula appears dark on FFA)



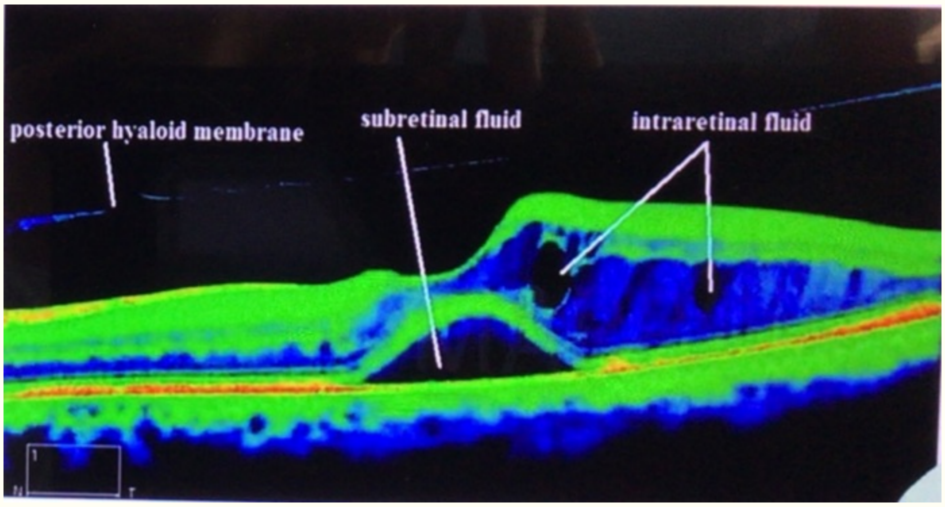
Leakage /Pooling of dye at macula : macular disease

3. Indo-CyanineGreen Angiography (ICGA)
- Dye : Indo-CyanineGreen ✓
- Better for visualisation of choroidal circulation as :
  1. It is 98% protein bound : Better retention
  2. Fluoresces at 810nm : Better penetration * ↑λ
- Contraindication : Iodine hypersensitivity *

4. Optical coherence tomography (OCT)
- It shows the layers.



- RPE : It is red in colour
- Fovea appears like a depression
- Foveola (Centre of Fovea) : GCL is absent. (Ganglion cell layer)

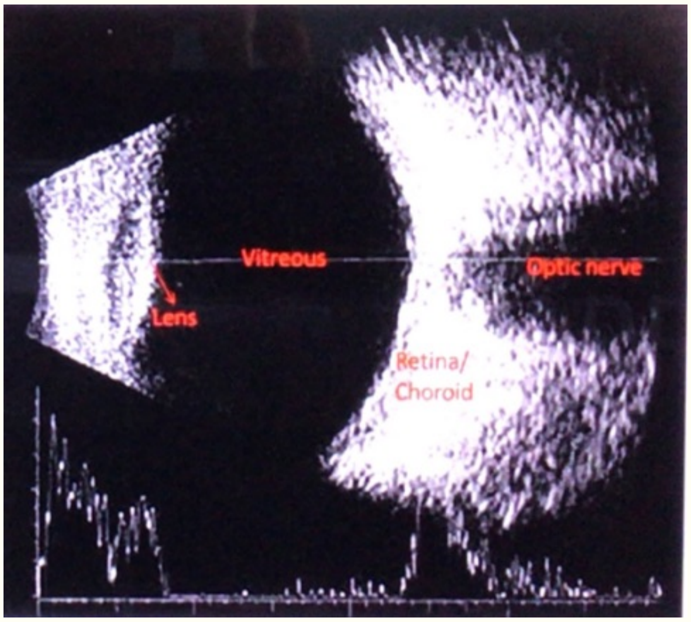


Investigations : B-Scan USG and ERG

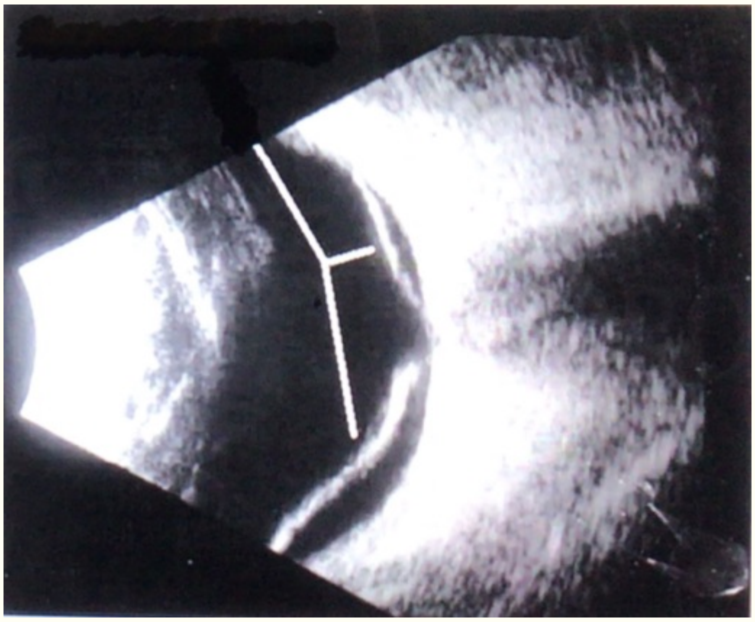
00:58:58

- Brightness scan
- 7.5 to 10 MHz probe

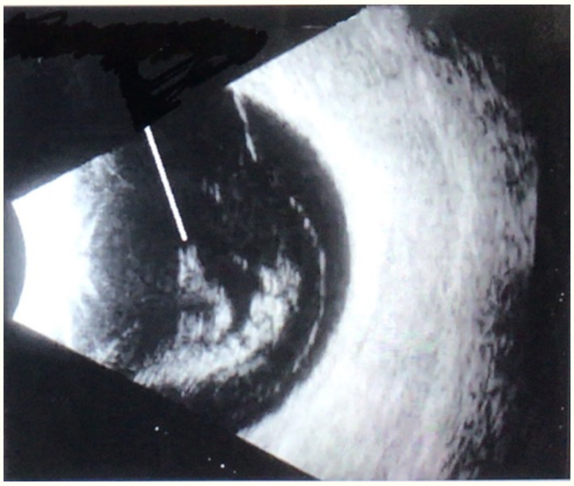
⇒ in pts c̄ ocular media opacity
(Corneal opacity)
or
(complete cataract)



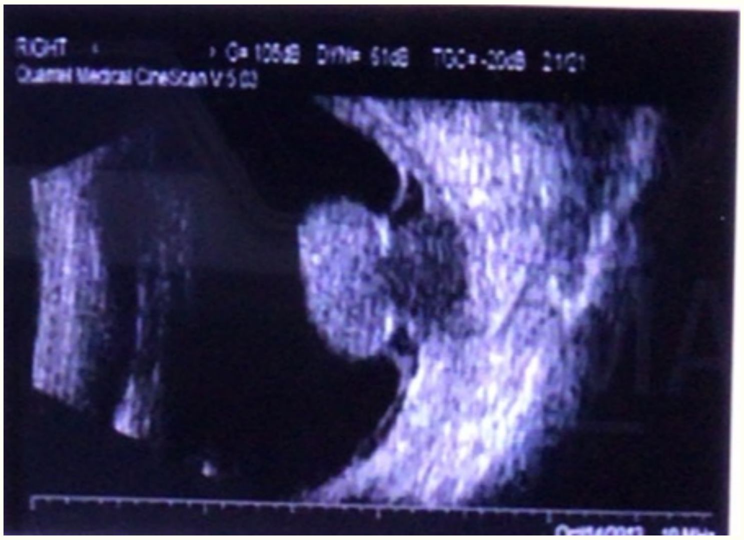
Normal B-Scan



Retinal detachment.



Vitreous hemorrhage

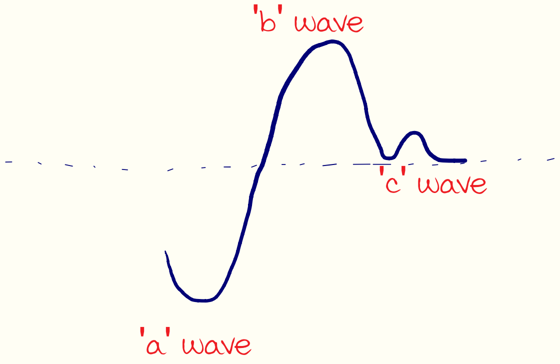

RIGHT
G= 105dB DYN= 51dB TGC=-20dB 21/01
Quantel Medical CineScan V 5.03

Choroidal Melanoma (mushroom shaped tumor)

- Growth of white area into vitreous : Retinal tumor – Retinoblastoma
  Choroidal tumor – Choroidal melanoma

6. Electroretinography (ERG)

- A record of action potential.
- Source : Panretina



- 'a' wave : Generated by Photoreceptors.
- 'b' wave : Generated by muller cells.
  Conducted by Bipolar cells.
- 'c' wave : metabolism in Retinal pigment epithelium.

Special ERGs :

a. Early Receptor Potential (ERP)
b. multifocal ERG

} Generated by Cones alone. *
Macular fn

c. Pattern ERG : Used to diagnose Glaucoma as it records responses from Ganglion cells, correlating with Optic nerve function.

Active space

Electrooculography

01:11:28

A record of standing potential.

Source :

a. Photoreceptors
b. Retinal pigment epithelium

Outer Retina

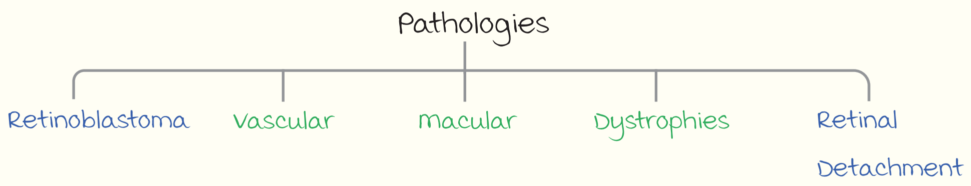
Responses are measured as Arden's ratio :

- Arden's ratio = $\frac{\text{Light peak}}{\text{Dark trough}}$
  Light peak → highest Recorded ↓ bright light Condns
  Dark trough → smallest value ∴ ↓ dark
- Normally Arden's ratio > 1.85 or 185%

RETINA: RETINOBLASTOMA, MACULAR DISEASES

Pathologies of Retina

00:00:07

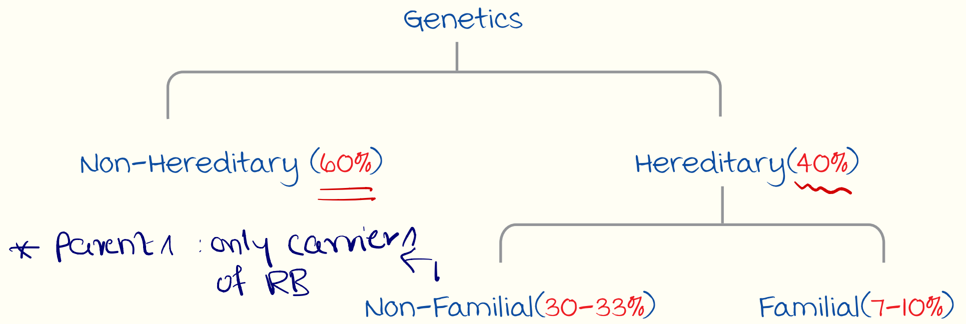


Vascular, macular pathologies and Dystrophies are groups of disorders while the other 2 are diseases..

Retinoblastoma[RB]

00:01:07

- Most common primary intraocular malignancy in children
- Most common age of presentation is <3 years. *



* Parent 1 : only carrier of RB

- Sporadic = Non-Hereditary+Non-Familial=90-93%.
- All Non-Hereditary cases are unilateral.

↳ family H/o ⊕

Hereditary Retinoblastoma

- Most common mode of inheritance: Autosomal Dominant
- Mutation of RB gene 13q14 (RB gene is a Tumour Suppressor Gene)
- ⇒ Knudson's 2 Hit hypothesis :
  - Inactive allele of RB gene is inherited ✓ → enough to cause tumor
  - 2nd mutation is acquired
  - 2nd mutation is *needed for Retinoblastoma to occur phenotypically *

- So Genotypically RB is Autosomal Dominant
- But Phenotypically it is Autosomal Recessive

- It states that there is loss of heterozygosity on and mutation that leads to presentation of RB

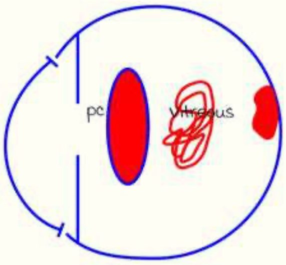
  - most common associated malignancy with RB: Pinealoblastoma (Primitive Neuro-endocrine Tumour [PNET])
  - most common associated secondary malignancy with RB : Osteosarcoma

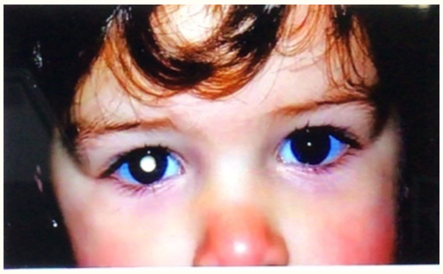
Clinical features of Retinoblastoma

00:08:58

- Clinical feature of RB :

1. most common sign: Leukocoria (White Pupillary Reflex)





Leukocoria

- most common cause of Leukocoria Unilaterally : RB
- most commoncause of Leukocoria Bilaterally : Cataract

2. and most common sign: Strabismus/squint [Esotropia (inward deviation)]
3. 3rd most common sign: Secondary glaucoma with buphthalmos (as age < 3y)

* most common route of spread: Direct spread (occurs through optic nerve)

* Growth Pattern :

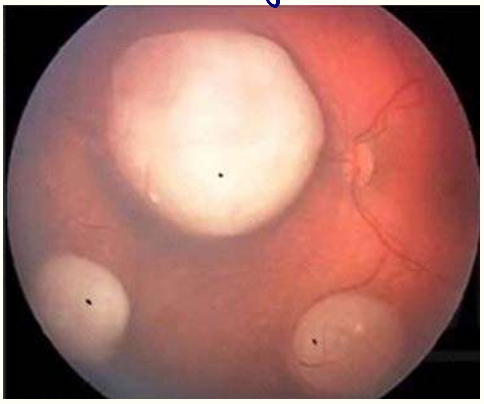
- Exophytic- growing towards Choroid

  ↓

- Retrolaminar growth : Tumour is growing behind Lamina Cribrosa
  : Poor prognosis✓
- Endophytic - growing in the vitreous cavity
  - examined on indirect ophthalmoscopy.

Active space

Endophytic



on Indirect opthalmoscopy

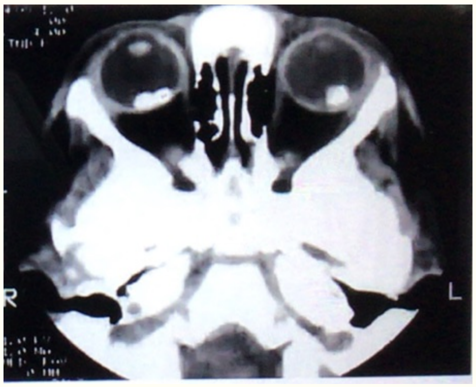
Investigation of Retinoblastoma

00:17:29

- Investigation of RB :
    1. Investigation of choice: CT scan

↓

As, RB is the MC cause of intraocular calcification



2. MRI : to assess the metastasis

⇒ 3. ↑LDH in aqueous (alt ↑met by tumor)

4. Gold Standard : Biopsy

↓

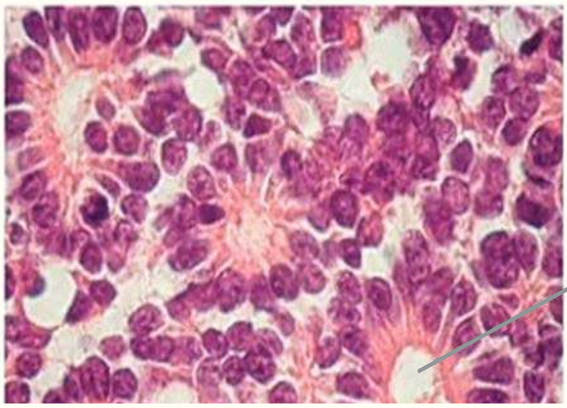
Done after removal of the whole eye

↓

⇒ Sample taken from optic nerve ⇐

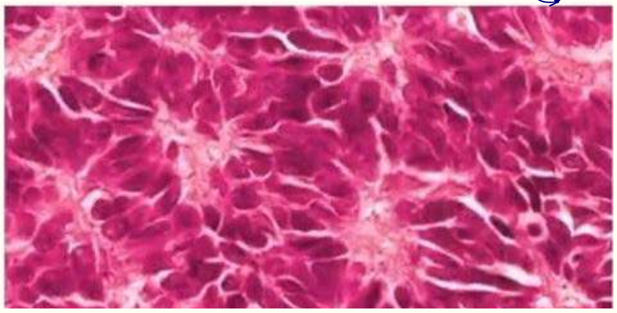
Histopathology Features :

a. Flexner-Wintersteiner Rosettes



Central empty lumen surrounded by columnar cells

b. Homer-Wright rosettes (pseudo-rosettes)



No central lumen

c. Fleurettes

Treatment of Retinoblastoma

00:22:53

- Treatment of RB

1) ENucleation : Removal of whole eye ball along $\bar{c}$ O.N

2) Chemotherapy – C – Carboplatin
E – Etoposide
V – Vincristine

3) U/L RB Gr 2 : C, V
3, 4 → CEV
5 → ENucleation

Grade 5 : (if any one of these 3 conditions is present)

i. Tumour occupies >50% of total volume of eye
ii. If vitreous hemorrhage is present
iii. If Rubeosis iridis seen (neovascularization of iris)

Active space

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Macular Function Tests

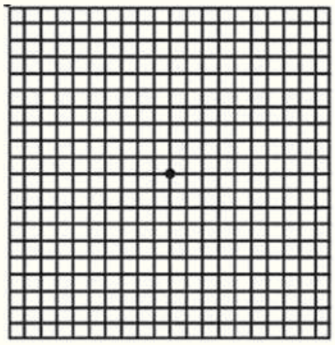
00:27:39

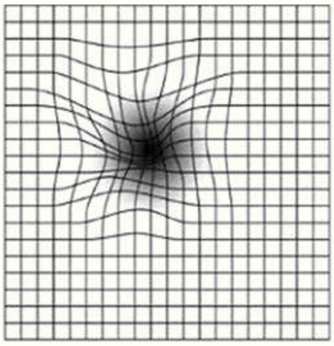
Macular Function Test

Clear media
- ✓ 1. Visual Acuity measurement
- ✓ 2. FFA
- ✓ 3. OCT
- NEET 2020 ★ 4. Photostress test:
  Shine light on retina for 10 sec
  ↓
  Record time of recovery of vision * to within 1 line of pretest vision * (ex: 6/12 → 6/18)
  - Time taken normal – Optic nerve disease
  - Time taken more – macular disease ✓

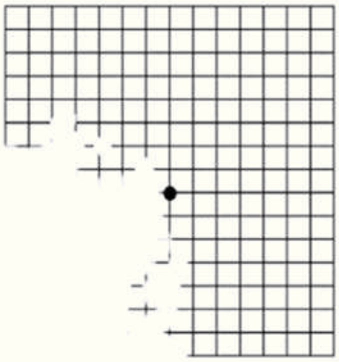
Hazy media
- 1. Maddox Rod Test ✓
- 2. multifocal ERG (only from cones)
- 3. LI (Laser interferometry)
- 4. PAM (Potential Acuity meter)
- 5. Entoptic Phenomenon

5. Amsler's grid: Square chart with 400, 5*5mm squares


a) Ⓝ person


wavy lines

b) macular d/s


c) Optic nerve disease (missing lines)

Macular disorders: Central Serous Retinopathy and Cystoid Macular Edema

00:35:02

CSR Central Serous Retinopathy	CME Cystoid macular Edema - Collection in outer plexiform layer (6th layer)
Collection of fluid at macula in subretinal space Pathology defect: Retinal Pigment Epithelium (RPE) Risk factors : 1) males(25-55 years) 2) Type A personality : workaholic prone to stress 3) Steroids 4) Cushing's syndrome 5) H. Pylori infection ✗	Risk factors : R : Retinitis Pigmentosa U : Uveitis N : Niacin/nicotine For P : Prostaglandin analogues R : Retinal vein occlusion I : Irvin Gass Syndrome (Cataract Sx complication) D : Diabetic retinopathy E : Epinephrine in aphakia
Clinical features: A. Loss of central vision B. metamorphopsia C. OCT : ✓ • Loss of foveal depression • Shallow neurosensory reti detachment in chronic cases D. On FFA (Pattern of leakage or pooling of dye) 1. Ink blot appearance 2. Smoke Stack Appearance or Umbrella appearance	Clinical features: A. Loss of central vision B. metamorphopsia C. OCT : ✓ • Loss of foveal depression • Cystic spaces in OPL in chronic cases ✓ D. On FFA (Pattern of leakage or pooling of dye) 1. Flower petal Appearance or Petaloid appearance
RIF ← 1) Steroids are contraindicated ✓ 2) most cases are self-resolving 3) If CSR persistent for >4months Or If recurrence ✓ Or If visual field defect in other eye ✓ ↓ A. Laser photocoagulation → fibrosis B. Photodynamic therapy (PDT) : uses a drug called "verteporfin" then shine light of 689nm for drug to start acting.	Treatment : 1) Steroids are mainstay (leakage ∵ Inflammⁿ)

Active space

Following pdfs notes are available free→
1→marrow edition 4 notes(latest optha notes also available) , marrow qbank , marrow pearls
2-> prepladder notes, prep rapid revision 2.0,prepladder treasures, prepladder snapshots
3-> Notespaedia devesh mishra pathology notes, notespaedia marrow Ecg, Notespaedia elite optha, notespaedia Dilip sir medicine notes, notespaedia micro pure, notespaedia pharma pure, notespaedia ent pure
4-> egurukul - - >aswini Kumar anatomy notes, dermatology notes, medicine by thameem sir, ortho notes, physio notes, radio notes(all by notespaedia)
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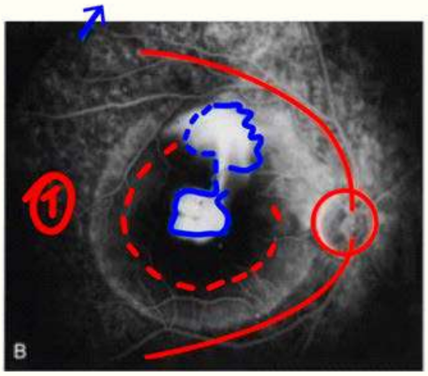
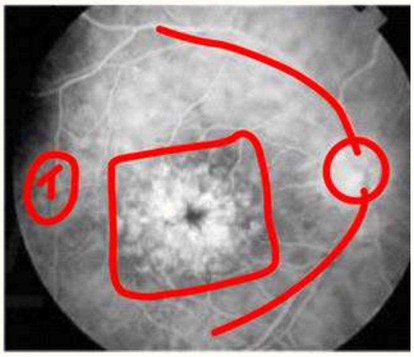
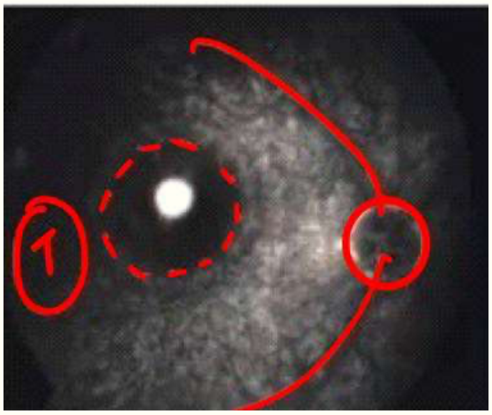
a) SSA/UA

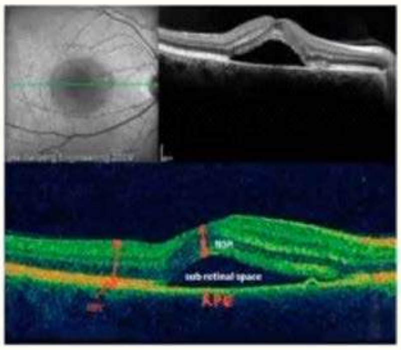


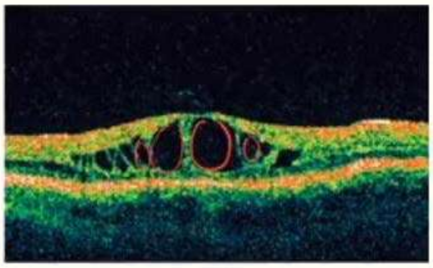
b) Flower appearance





Ink blot





CME

MOP
↓ ↳ outerplexiform
CME

CSR
↳ Sub Retinal space

Age Related Macular Degeneration [ARMD]

00:54:35

- Risk Factors:

i. > 50years.
ii. Smoking
iii. Blue iris *
→ iv. High fat diet

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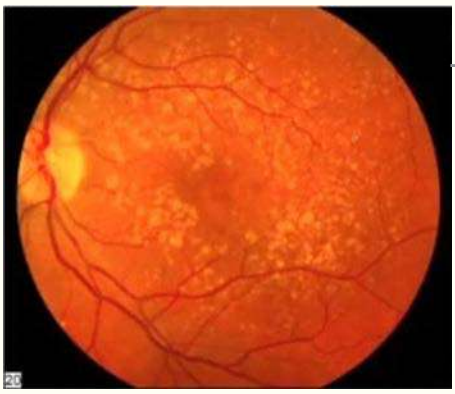
Types

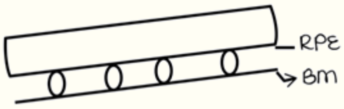
- Dry / Non - exudative
  - Early
  - Intermediate
    - (Early and Intermediate) i. Drusen
    - ii. Hyperpigmentation Of RPE ×
  - Late
    - Geographic atrophy of RPE
- Wet / Exudative
  - PED
  - CNV (Choroidal Neovascularisation

Drusen : extracellular eosinophilic deposit between RPE and Bruch's membrane. ☆

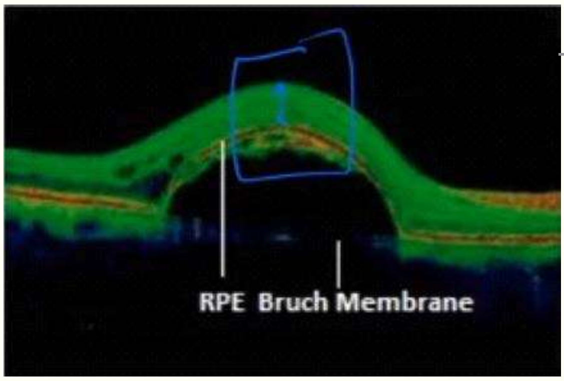
They are below Retinal B/vs
↓
i'e below Retina



→ Drusen

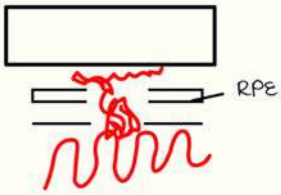


PED : Pigment epithelium detachment
↓



→ PED

drusen
↓
RPE, choroidal ep
atrophy



CNV
↓
Subret H'ge
↓
Fibrovasc. complex formed
(Choroidal NV memb)

Active space

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CNV: Choroidal Neovascularization

01:00:40

CNV : Choroidal Neovascularization

↓

Grow into subretinal space leading to hemorrhage

↓

Fibrovascular complex forms

Known as CNVM (Choroidal Neovascular membrane)

Occult CNVM
In sub RPE space
IOC : ICGA ✶
(Choroid)

Classical CNVM
formed in subretinal space
IOC : FFA ✶

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

- Treatment:

Dry	Wet
Antioxidants CAREDS 2 study ↓ Vit. C & E ⊕ Zn & Cu ⊕ Lutein & Zea-xanthin (Caretenoids)	TOC : Anti VEGF (Intravitreal) i) Bevacizumab ii) Ranibizumab → ⊖ VEGF-A iii) Aflibercept → soluble decoy ® iv) Brolucizumab ★ (new) Photodynamic therapy Laser photocoagulation ↓ if extrafoveal CNVM

→ for subfoveal or Juxtafoveal CNVM

Active space

Other Macular Disorders

01:09:44

- Epimacular membrane: also known as cellophane maculopathy
- Angioid Streaks
  - Cracks in Bruch's membrane
  - Causes

    P : Pseudoxanthoma elasticum
    E : Ehler Danlos syndrome
    P : Paget's disease
    S : Sickle cell disease
    I : Idiopathic

- Hypotony maculopathy :
  - Develops when IOP <5mm Hg
  - Causes :

    i. Excessive drainage after glaucoma surgery
    ii. Retinal detachment → ↓IOP
    iii. Penetrating trauma ✓
    → iv. Chronic uveitis ✓ : ciliary body malf

Active space

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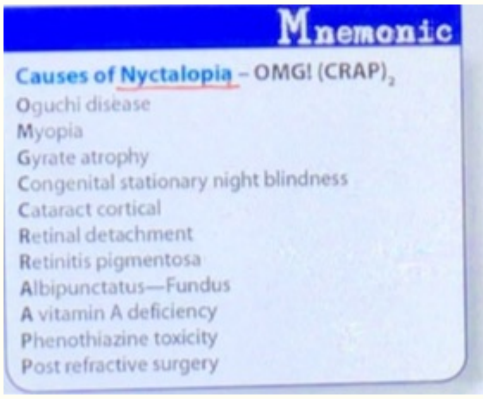
RETINA: FUNDUS DYSTROPHIES

Introduction

00:00:16

Dystrophies which affect photoreceptor retinal pigment epithelium choriocapillaris complex and lead to rod & cone degeneration.

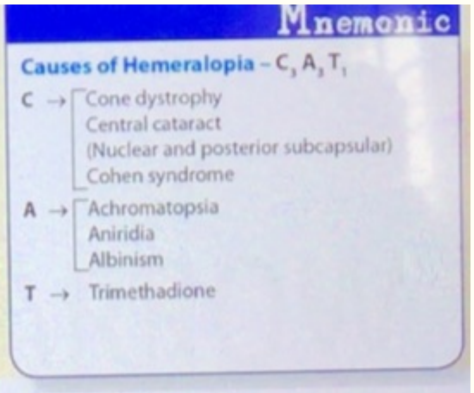
Rods	cones
Night vision (scotopic vision)	Colour/day vision (photopic)
Peripheral vision, as rods are maximum in perifoveal area	Central vision, as cones are maximum at fovea
Lesion – Nyctalopia (night blindness) Peripheral loss of vision	Lesion – Hemeralopia (colour/dayblindness) Central scotoma



Mnemonic

Causes of Nyctalopia – OMG! (CRAP)$_2$

Oguchi disease
Myopia
Gyrate atrophy
Congenital stationary night blindness
Cataract cortical
Retinal detachment
Retinitis pigmentosa
Albipunctatus—Fundus
A vitamin A deficiency
Phenothiazine toxicity
Post refractive surgery



Mnemonic

Causes of Hemeralopia – C$_3$, A$_3$, T$_1$

C → Cone dystrophy
Central cataract
(Nuclear and posterior subcapsular)
Cohen syndrome
A → Achromatopsia
Aniridia
Albinism
T → Trimethadione

Retinitis pigmentosa

: mc heriditary fundus dystrophy

00:07:53

- Autosomal recessive
- Autosomal dominant – best prognosis
- X linked recessive – worst prognosis

Active space

Symptoms :

Rods > cones

* It is a rod - cone dystrophy. Initially, rods are affected and later cones.

mid peripheral superior scotoma

↓

Ring scotoma or doughnut scotoma (central vision preserved and peripheral vision loss)

↓

Tubular or tunnel vision ✓

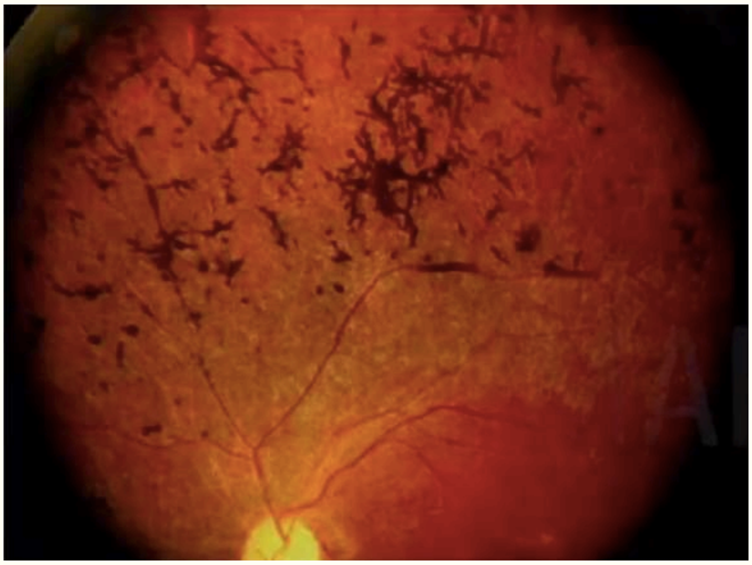
Signs of retinitis pigmentosa

00:13:35

Diag sign :-

1. Arteriolar attenuation
2. Optic disc- waxy, pale (consecutive optic atrophy)
3. Bony spicule pigmentation (perivascular)
4. Shining reflex at macula known as tapetal reflex - Only in X linked recessive female carriers.

Triad



Pigments in periphery

Arterior Attenuation

Pale OD

Retinitis Pigmnetosa - Bone Spicule Pigmentation

Diagnosis

IOC - ERG (Electroretinography)

Treatment of retinitis pigmentosa 00:17:54

In India - no treatment

The possible treatment-
- ARGUS II retinal prosthesis - Restores only central vision. *
- Voretigeneneparvovec - Used if biallelic RPE65 gene mutation. It is adeno associated virus vector based gene therapy. *

Systemic associations of retinitis pigmentosa 00:21:14

Lawrence moon biedl syndrome - most common
- Retinitis pigmentosa
- mental retardation
- Obese
- polydactyly

Usher's syndrome
- Retinitis pigmentosa
- Deafness

Refsum disease
- retinitis pigmentosa
- Cerebellar ataxia
- Polyneuropathy

Bassenkornzweig syndrome
- retinitis pigmentosa
- Abetalipoproteinemia
- Acanthocytosis (spur cells)
  ↓
  PBS

Active space

Cones
↑

Best's macular dystrophy

00:24:34

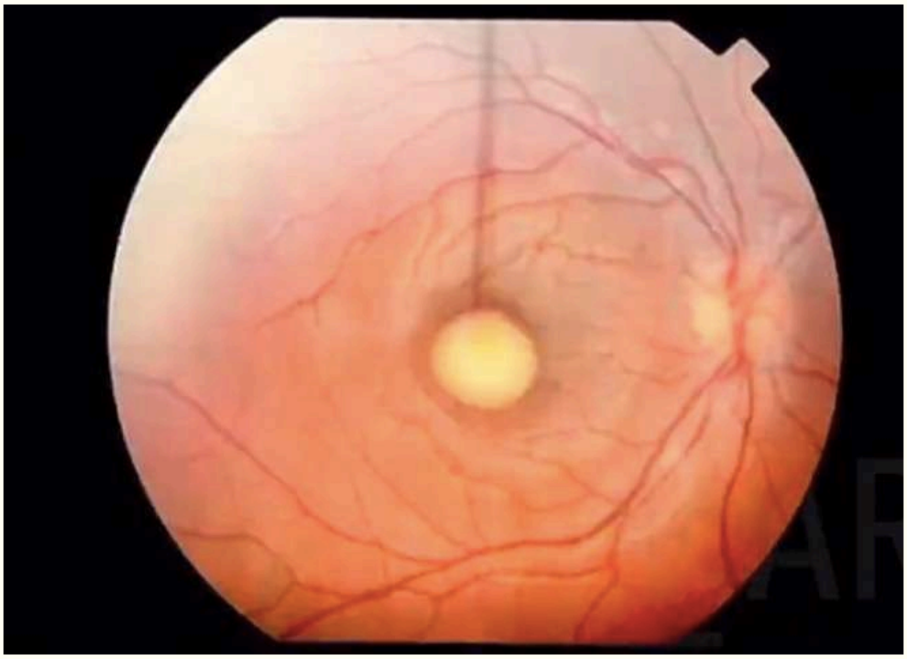
It is autosomal dominant

Symptoms :

It is primarily a cone dystrophy ✗
C/F – hemeralopia (colour/day blindness) ✓
Central scotoma

Signs :

1. Lipofuscin accumulation
2. Fundus appearance – vitelliform lesion (egg yolk like)
3. Pseudohypopyon – vitelli eruptive stage



Diagnosis

AI 2011 ☆

Active space

IOC : EOG* – measured by arden's ratio <1.50 or 150 %
→ ERG is normal.

Stargardt's disease

00:29:56

It is autosomal recessive

MC juvenile macular dystrophy - cone dystrophy.

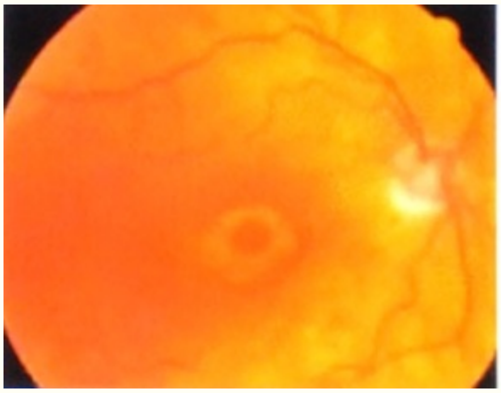
C/F - Hemeralopia (colour/day blindness) ✓

Central scotoma. ✓

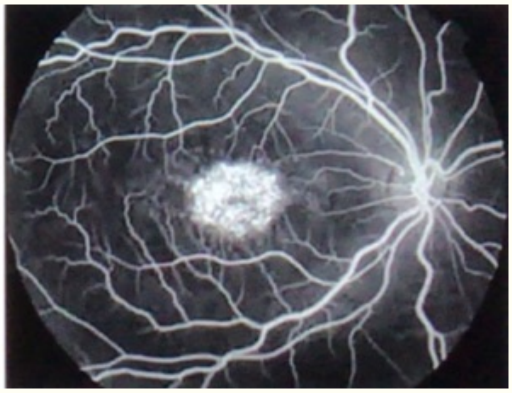
Signs :

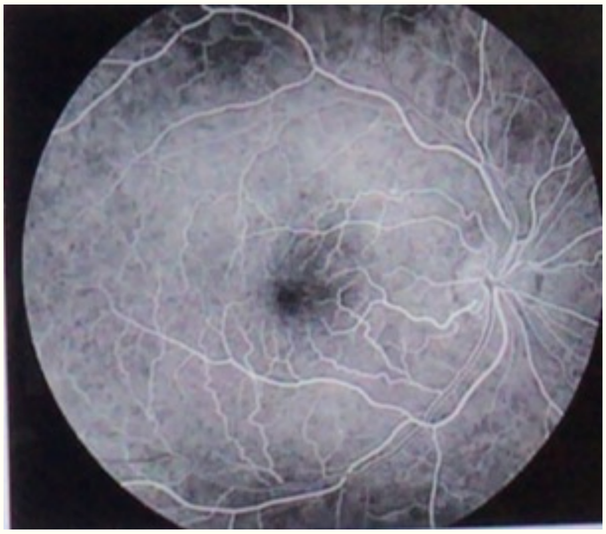
1. Bull's eye maculopathy
2. On FFA - dark choroid sign or silent choroid sign

Bull's eye maculopathy



Dark choroid sign or silent choroid sign





Normal FFA - background fluorescence due to dye in choroidal vessels *

Bull's eye maculopathy 00:34:30

It is due to chloroquine toxicity

Stages :

1. Pre maculopathy – It is reversible, there is normal vision with central scotoma
2. maculopathy – Bull's eye lesion seen, it is irreversible and decreased visual acuity.

   It occurs if: Daily dose >3.5 mg/kg or 250 mg ✓
   Cumulative dose >460 mg
   Duration of treatment > 5 years
   Renal or hepatic function compromise.

Chloroquine can also lead to vortex keratopathy.

Congenital night blindness disorders 00:38:12

It presents with infantile onset nyctalopia

Disease	ERG	Fundus
Congenital stationary night blindness	–ve (No ⓑ wave)	Ⓝ *
Oguchi blindness	Ⓝ *	Abn.
Enhanced S – cone syndrome	–ve	Abn.
Fundus albipunctatus	Absent Rod response	Abn.

Oguchi blindness

- There is no damage to rod cells, instead there is damage in conduction.
- Therefore, it is diagnosed by the mizuonakamura phenomenon ★★★★
  – in light conditions fundus has golden yellow colour.

Miscellaneous conditions

- X linked retinoschisis (schisis – split)
- There is split in retina between nerve fibre layer and rest of retina.

  * It presents with bicycle wheels maculopathy. *
- Stickller's syndrome – * MC inherited cause of RD in children. *

Active space

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RETINA: VASCULAR DISEASES

Vascular retinal disorders are group of disorders that affect the blood vessels of retina.

Diabetic retinopathy (DR) 00:00:55

- Risk factors :

  Duration of diabetes is directly proportional to risk.

Screening for DR

IN T1DM → After 5 years

IN T2DM → Immediately upon diagnosis ✓

- Increased HbA1c.
- HTN and hyperlipidaemia.
- Pregnancy.
- Diabetic nephropathy → serum creatinine relates with risk for diabetic retinopathy.

* Sr Creat levels *

Pathogenesis of DR :

Biochemical mechanisms of cellular damage :

1. Intracellular sorbitol accumulation.
2. Accumulation of ROI's (reactive oxygen intermediates).
3. PKC (protein kinase-C) activation. ✓
4. Advanced glycation end-products formed

↓

Anatomic lesions :

Loss of pericytes (covering of contractile cells around capillary endothelium to prevent damage) is the earliest histologic change.

↓

Leakage and occlusion of capillaries.

↓ Hypoxia ← CRAO CRVO

Hypoxia

Release of VEGF (vascular endothelial growth factor).

↓

Neovascularization (abnormal leaky vessels)

Clinical features of DR :

00:13:17

ETDRS (early treatment diabetic retinopathy study) classification :

NPDR (Non proliferative diabetic retinopathy)

PDR (Proliferative diabetic retinopathy)

↓

NV

NVD

NVE

o/e : Tortuous tuft of v/s = seen

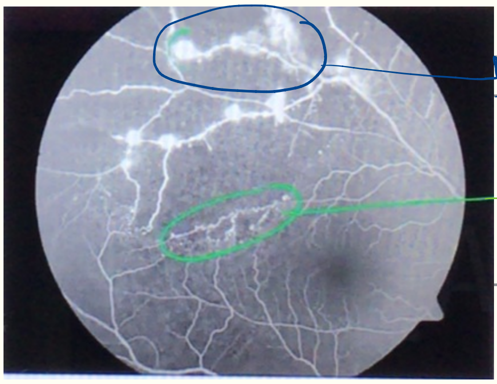
MAIN

NPDR (non proliferative DR) :

1. Microaneurysm :
   - Earliest sign
   - O/E : red dot like lesions.
   - On FFA : hyperfluorescent small dots. ←
   - Layer involved : INL (inner nuclear layer).

FFA

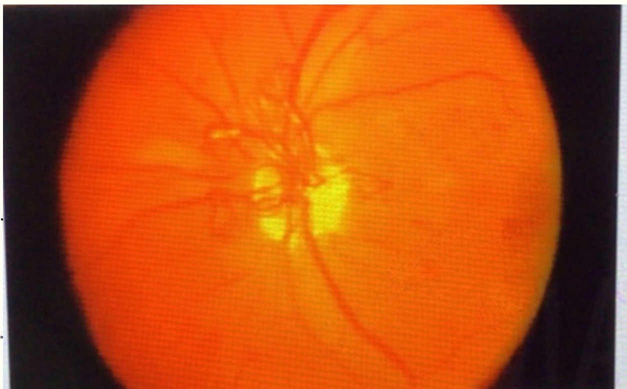
↓



Neovascularization

Pooling of dye – due to neovascularization

Hyperfluoroscent spots – microaneurysms



2. Haemorrhages :

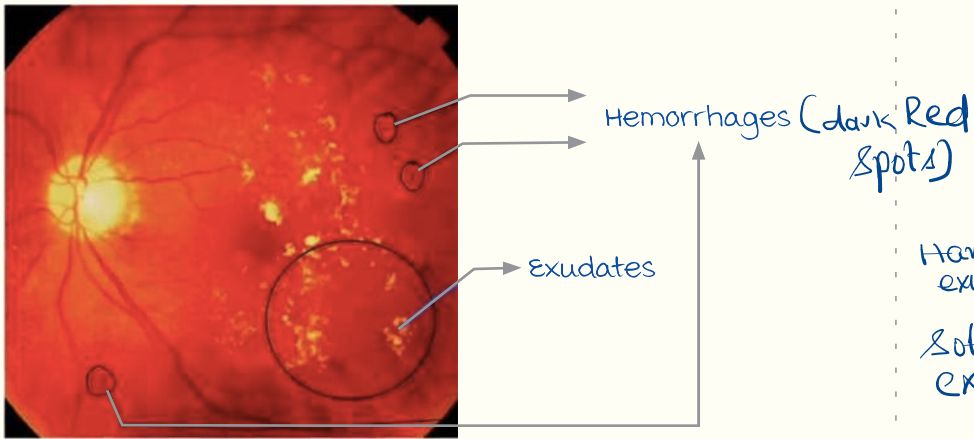
Flame shaped | Dot and blot
Superficial | Deep
Involves nerve fibre layer | Involves outer plexiform layer

3. Exudates :

Lipid deposition involving outer plexiform layer

O/E : yellow irregular shaped deposits.

On FFA : hypo fluorescent spot.



Hard exudate → Exudate

Soft exudate → Cotton wool spots

4. Cotton wool spot :

Collection of neuronal debris.

Seen on nerve fibre layer.

O/E : dirty or, pale yellow large spots. *

On FFA : hypofluorescent area.

5. Venous beading/looping.

6. IRMA (intraretinal microvascular abnormalities) – it's a shunt vessel.

Active space

Treatment of DR : 00:29:12

NPDR

Glycaemic control and follow up.

Stage	Follow up every
mild	9-12 months
moderate	6 months
Severe	4 months
very severe	2 months

PDR

1. Treatment of choice - PRP (pan retinal photocoagulation) using argon or, frequency doubled Nd : YAG laser, in multiple sittings. Ⓡ

   S/E of PRP : visual field defects.

2. Anti VEGF drugs

↓ O_2 demand
↓ leakage
↓
but if PC occurs deeper & involves Rods & Cones
↓
VF defects

Note : - PDR treatment is done only if :

a. NVD = 1/3 of disc area.
b. NVD with vitreous haemorrhage.
c. NVD >or = 1/2 of disc area with vitreous haemorrhage.

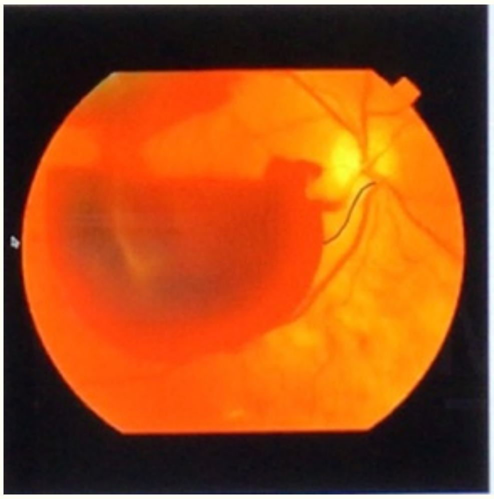
Complications of DR 00:36:58

- most common cause of loss of vision in DR - macular edema.
- most common cause of loss of vision in NPDR - macular edema.
- most common cause of loss of vision PDR - Vitreous haemorrhage.
- ⇒ most common cause of blindness in DR - Retinal detachment.

Vitreous haemorrhage :

1. Source - neovascularization of retina.
2. most common cause of vitreous haemorrhage - DR (diabetic retinopathy).
3. most common cause of vitreous haemorrhage in young adults - trauma
4. most common cause of recurrent vitreous haemorrhage - Eale's disease.
5. Treatment - endophotocoagulation → pars plana vitrectomy

Active space



Boat shaped vitreous haemorrhage

Macular edema (in diabetes) :

00:42:21

1. Occurs due to leakage.
2. Treatment :
   - Focal laser photocoagulation. ✓
   - If centre involving macular edema/foveal edema – Anti VEGF drugs.

⇒ Intravitreal triamicinilone injection.

↓

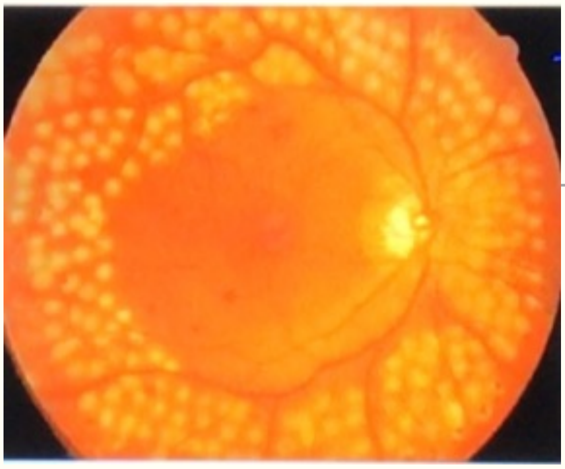
Preferred if :

a) Ischaemia patient. ✓

b) Pseudophakia. ✓

c) Patient having CVDs.

TIP(S)



PRP : spots

Telegram--@latestpgnotes

Hypertensive retinopathy (HTNR)

00:046:50

- vasocontrictive phase
  ↓
  Sclerotic phase (Atherosclerosis)
  ↓
  Exudative phase

*

a/t sclerosis & constriction

C/F: Keith-wagner classification

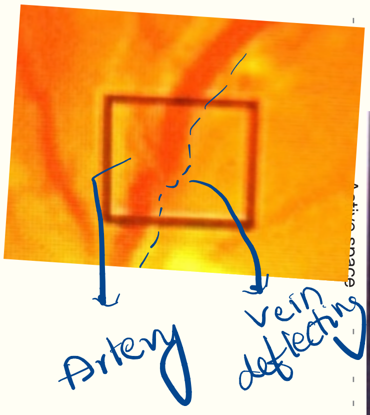
G1 : Arteriolar attenuation
(Ⓝ A : v ratio (diamter) = 2:3
HTN Retinopathy → 1:3)

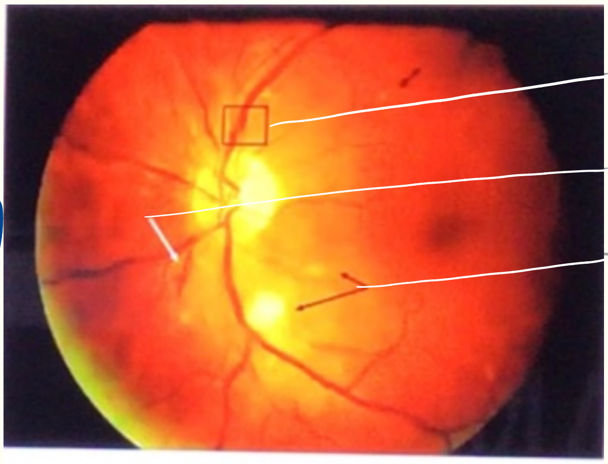
G2 : Salus sign : Deflection of B/v
G3 : Bonnet sign : Banking of B/v
Gunn sign : Tapering
→ seen @ AV Crossing
↓ seen in veins

(Rigid Artery compressing soft vein)

- Flame shaped H'ges c̄ cotton wool spots
- copperwiring of Arterioles

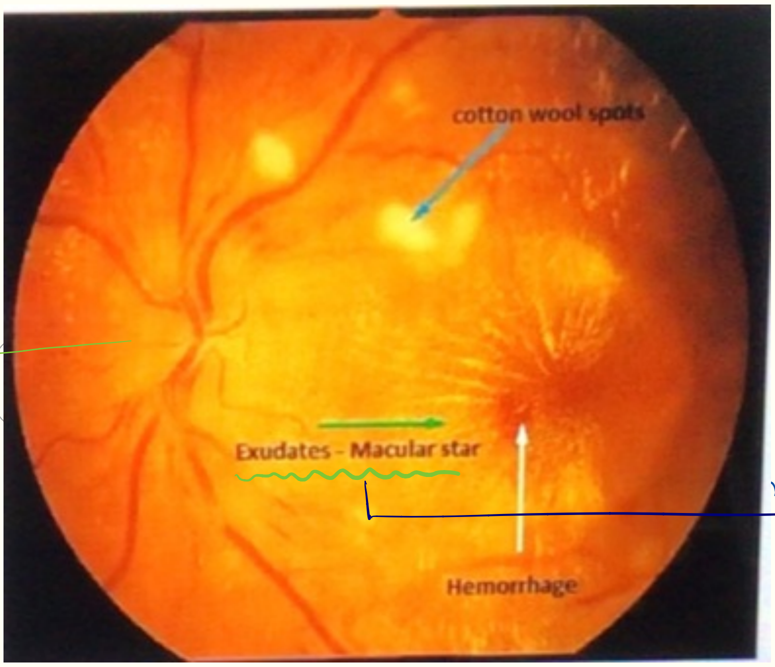
G4 : · silver wiring of Arterioles
· Papilloedema





Hypertensive Retinopathy

Gr-3



Gr. 4

Swelling of O.D

Seen in Malignant HTN

Central retinal artery occlusion (CRAO)

00:57:00

- Causes of CRAO :
  1. most common – atherosclerosis.
  2. Giant cell arteritis.
  3. Emboli – Hollenhorst plaque.
     ↓
     Cholesterol emboli.
     Originates from bifurcation of common carotid artery.

Clinical features of CRAO :

✓ 1. Sudden and painless loss of vision.
2. Optic atrophy – consecutive.
3. Pupil – RAPD or, marcus Gunn pupil. [Partial ON lesion]
4. Fundus appearance – pale fundus (due to retinal edema).
   -Cattle tracking fundus (due to segmentation of blood column).
5. Cherry red spot at macula – due to collection of fluid in ganglion cell layer of retina, which is absent at foveola making it transparent.

d/t No blood flow & contractile action of v/s-

GCL ↓ absent @ Foveola

Differential diagnosis of cherry red spot at macula : edema/Accum.

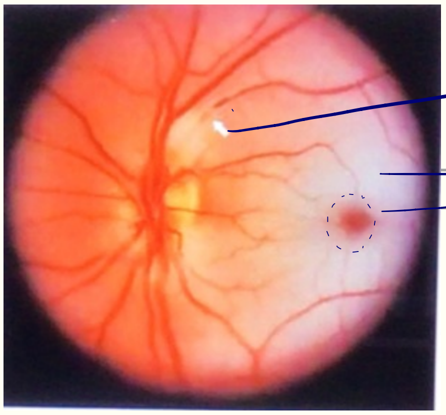
a) CRAO.
b) Trauma. (blunt)
c) Neimann Pick's disease.
d) Gangliosidosis – type I.

Cherry Trees Never Grow

Active space

Tall in Sand Mud & Grime

e) Tay Sach's disease. } → Gangliosidosis – type ②
f) Sandhoff's disease.
g) metachromatic leukodystrophy.
h) Gaucher's disease. — 1 – CRS ⊖ : m/c
2 – CRS ⊕
3 – CRS ⊕/⊖



No dye in any blood vessel
But, dye filling cilioretinal artery

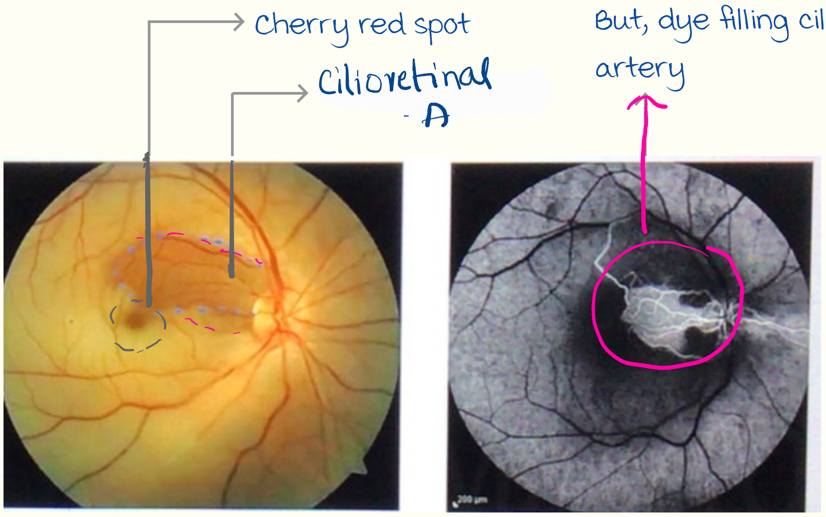


Fig:CRAO with macular cilioretinal artery spared with its corresponding FFA image.

Treatment of CRAO : 01:13:19

Treatment should be done within 4 hours (within 24–48hours occlusions become complete).

✓ 1. Ocular massage – dislodging emboli. : Compress – expansion
2. Vasodilatation :
⇒ Sublingual isosorbide nitrate. ✓
→ Carbogen inhalation (95%O2 + 5%CO2).
↓
↑ pCO2
↓
↓ pH
↓
vasodilatation

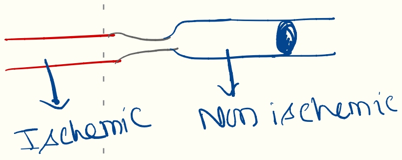
Active space

3. Lower the intraocular pressure : dilate b/v
   IV mannitol. ✓
   ☆ Paracentesis (aspiration of aqueous).

Central retinal vein occlusion (CRVO)

01:18:43

- Causes of CRVO :
  most common - hypertension. *
- Stages of CRVO :
  (Both stages are continuous, i.e. first non-ischemic stage occurs, then the ischemic stage)



CRVO
- Non-ischemic CRVO
- Ischemic CRVO

Non-ischemic CRVO :

a) Due to stasis of blood, leading to hypoxia and increased vascular permeability.

↓

macular edema ✓

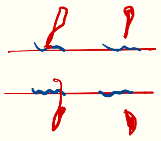
↓

Loss of vision

b) Treatment :

Intravitreal triamicinilone.
0.7mg dexamethasone intravitreal implants (GENEVA study).
Anti VEGF drugs.

Ischemic CRVO :



a) Due to severe hypoxia

↓

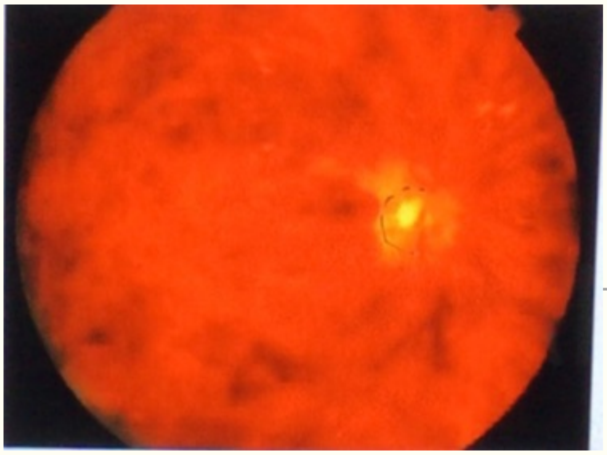
Damage of capillary endothelium,

↓

Clinical features :

1. Severe flame shaped haemorrhages
   Fundus – tomato Ketchup/ splash appearance.

Active space



→ Tomato ketchup appearance of fundus

2. Rubeosis iridis (neovascularization of iris)

↓

Occurs due to O2 demand by posterior segment

↓

Leaking of blood in anterior chamber

↓

Blocks trabecular meshwork ✓

↓

Causes neovascular glaucoma

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

Neovascular glaucoma in CRVO occurs after around 3months, known as 100-day glaucoma.

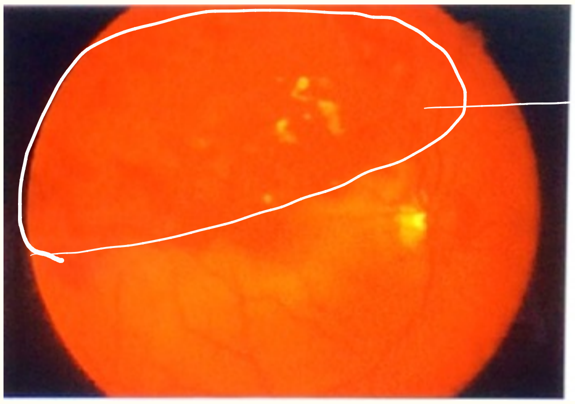
b) Treatment of ischemic CRVO :

⇒ PRP (pan retinal photocoagulation).

Branched retinal vein occlusion (BRVO)

01:30:00

- most common site of occlusion – AV crossings*
- most common quadrant affected – supreotemporal. ✓



→ Haemorrhages

Telegram--@latestpgnotes

Retinopathy of prematurity (ROP)

01:31:21

- Occur in premature child, when :
  Age at birth < or = 32weeks.
  Birth weight < or = 1750grams.

- Screening for ROP (4weeks after birth) :*
  Age at birth > or = 28weeks.
  and,
  Birth weight > or = 1200grams.

  Screening for ROP (2-3weeks after birth) :
  Age at birth < 28weeks.
  or,
  Birth weight < 1200grams.

28 —32

1200 —1750

or <28
<1200
↓
Screen

≥ 28 and
≥ 1200
↓
screen

- Signs/stages of ROP :

Stage 1 : Demarcating line b/w vascularised Retina & peripheral avasc.

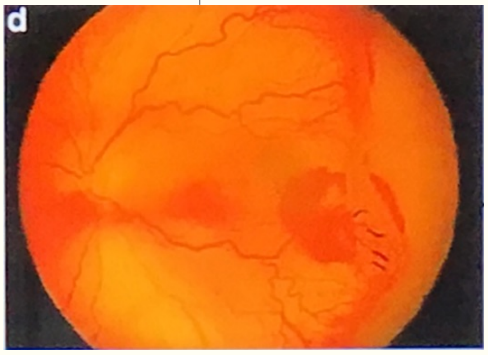
Stage 2 : Ridge

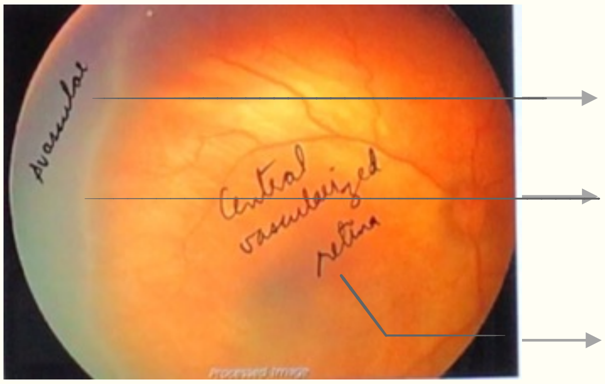
Stage 3 : ExtraRetinal NV on the Ridge moving into vitreous

Stage 4 : partial RD

Stage 5 : Total RD

Stage-3





Avascular part

Ridge

Central vascularized retina

- Treatment of ROP :
  a) Laser photocoagulation or, pars plana vitrectomy (if retinal detachment).

↓ done if pre-treshold ROP Type 1 (as told by ETROP study

Pretreshold ROP Type-1

· zone 1 affected ⊕ any stage ⊕ plus d/s

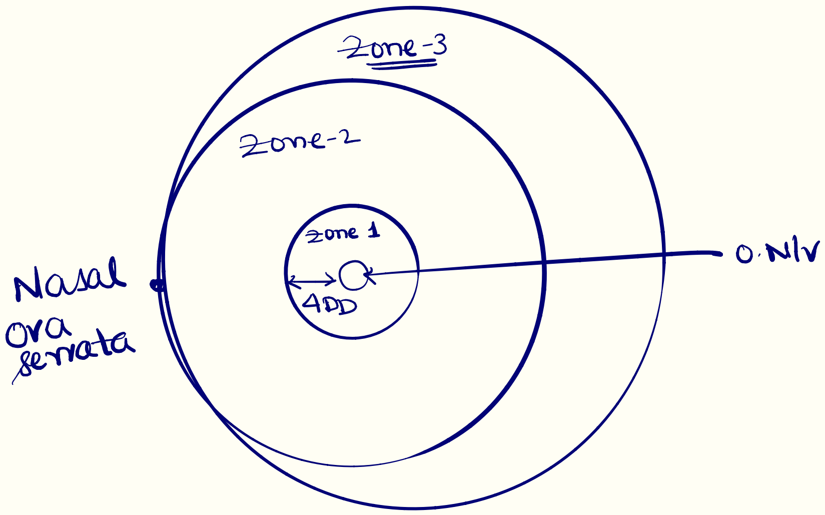
or

zone 1 ⊕ stage 3

or

zone2 ⊕ stage 2/3 ⊕ plus d/s

ROP → ROP plus d/s if venous tortuosity & dilatation in ≥2 quadrants



Eale's d/s

Triad - occlusion
periphlebitis
NV Retina : Rec. vitreous H'ge

Rx : PRP

Coat's d/s : Idiopathic Retinal Telangiectasia
m/c : ♂, always U/L

C/F : 1. m/c : painless LOV
2 Strabismus
3. Leucocoria
4. Exudative RD

RETINA: RETINAL DETACHMENT

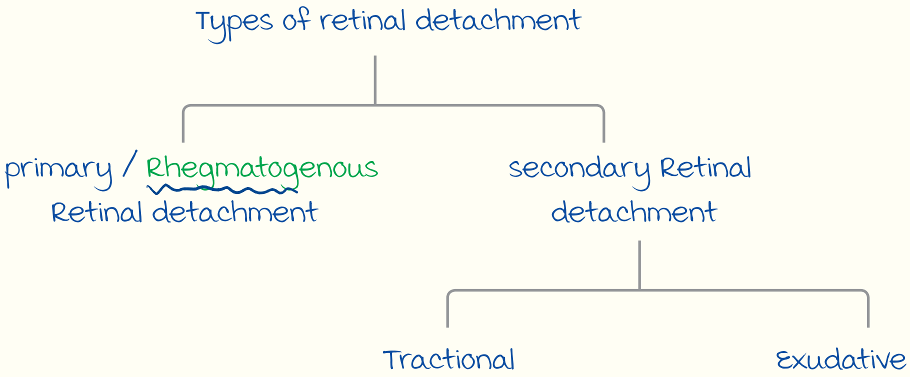
Introduction 00:00:06

It is the separation of the neurosensory retina from the retinal pigment epithelium and collection of subretinal fluid.

Investigations :

- Investigation of choice: Indirect ophthalmoscopy
- Optical Coherence Tomography (OCT)
- B-scan ultrasonography

Types of retinal detachment and primary retinal detachment 00:03:06


Types of retinal detachment
primary / Rhegmatogenous Retinal detachment
secondary Retinal detachment
Tractional
Exudative

Primary retinal detachment is also known as Rhegmatogenous retinal detachment.

'Rhegma' means a break (tear or hole) in the retina.

Pathogenesis :

- Synerесis (liquefaction of vitreous)

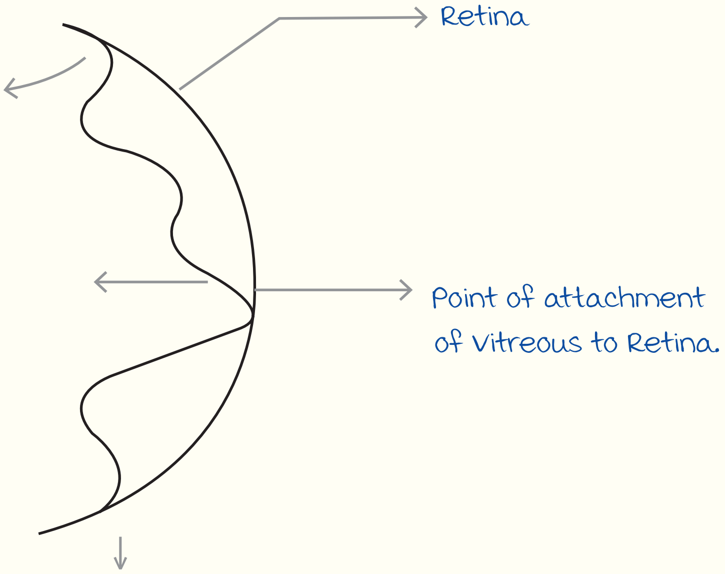
↓

Leads to posterior vitreous detachment (PVD)

↓

Some part of vitreous still attached exerts dynamic vitreoretinal traction.

Active space



↓

Retinal break develops

↓

The fluid enters through the break and seeps into the subretinal space

Causes :

1. most common : Pathological myopia
2. Blunt trauma
3. Cataract surgery (aphakia) ✓
4. Lattice degeneration of retina

five ectopic Neural impulse ⇒ Flashes

Symptoms :

1. Photopsia : Flashes of light ; Nlv fibres : stretched ⇒ irritated
2. Floaters : Small black flying dot like opacities.
   Due to vitreal opacities.
3. The patient complaints of curtain falling in front of the eye.

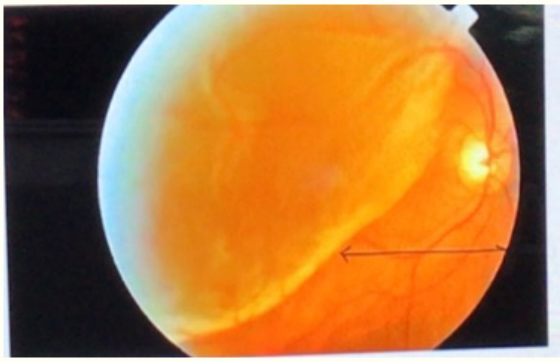
⇒ 4. Loss of vision : sudden and painless.

Primary retinal detachment : Signs and prophylaxis

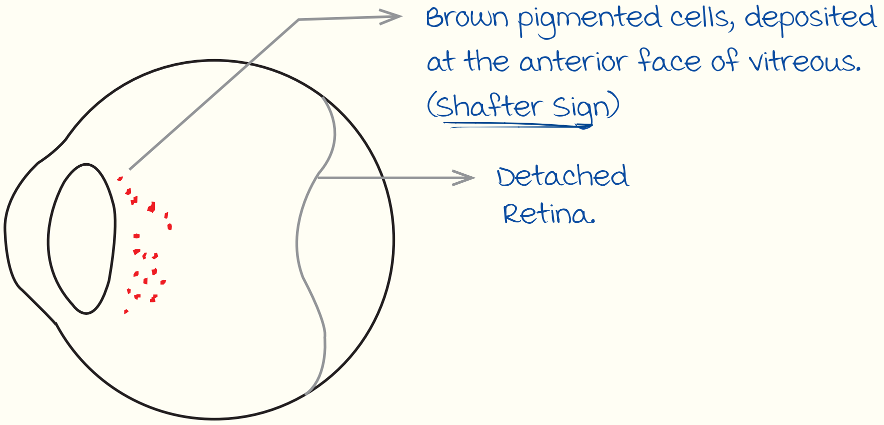
00:13:06

Signs :

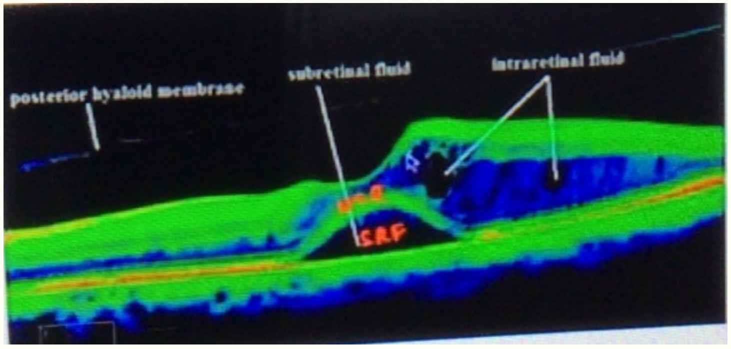
1. Detached retina : Gray/opaque, convex shaped



2. Retinal detachment : Extends till Ora serrata *
3. Shaffer sign : Tobacco dust appearance (alt vitreal changes)


Brown pigmented cells, deposited at the anterior face of vitreous. (Shafter Sign)
Detached Retina.

4. Relative afferent pupillary defect
5. In longstanding cases :
    - a. Retinal atrophy
    - b. Subretinal demarcation lines known as High water marks if (>3 months).
    - c. Intraretinal cysts (if > 1yr)



OCT image in long standing Rhegmatogenous retinal detachment.

Active space

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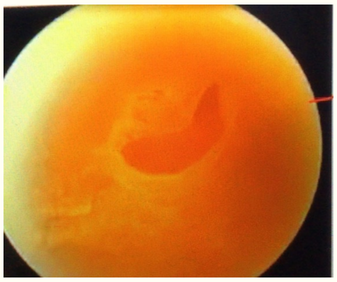
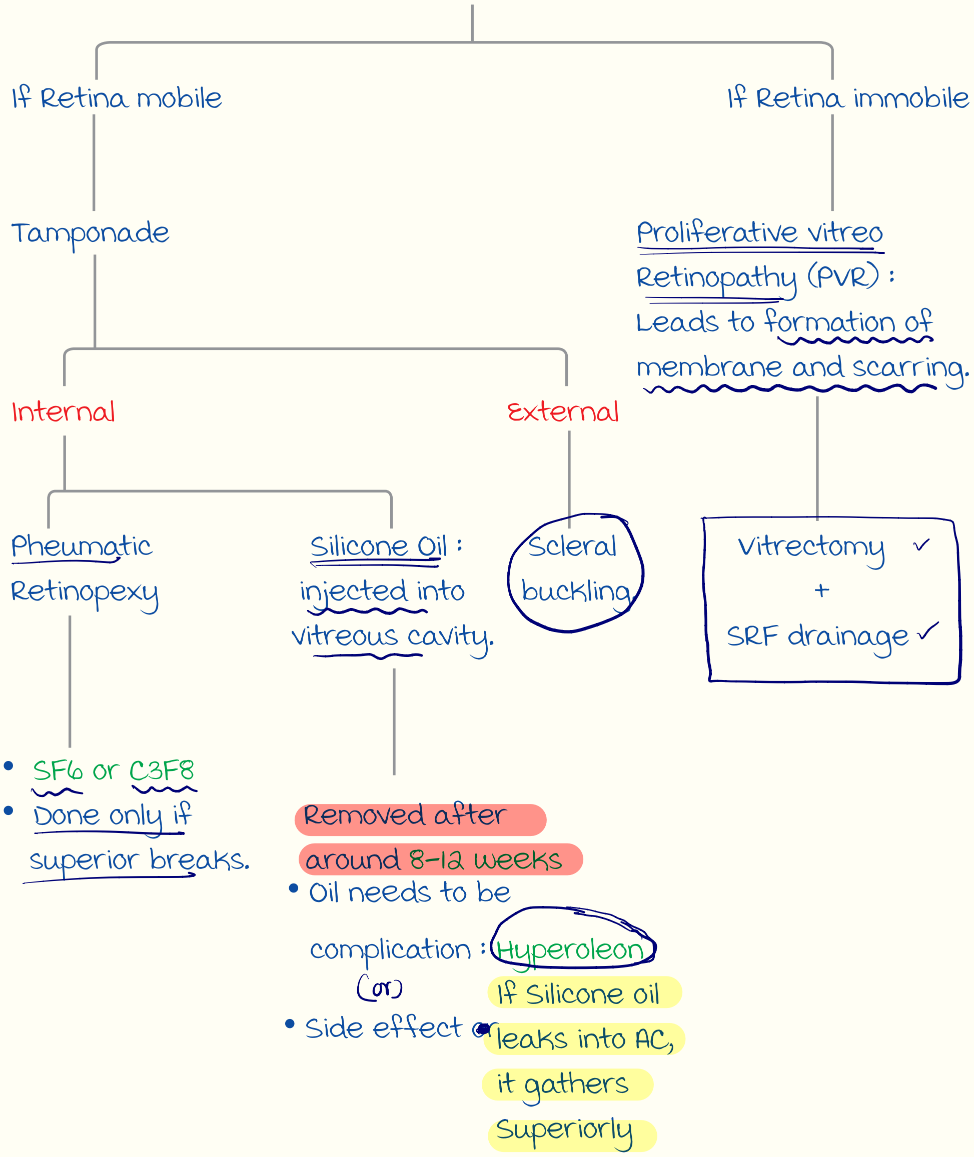


U shaped tear

Breaks that are most commonly seen in Superotemporal quadrant can be closed by laser photocoagulation.
(prophylactically)

Primary retinal detachment: Treatment

00:19:42



If Retina mobile

Tamponade

Internal

Pneumatic Retinopexy

- SF_6 or C_3F_8
- ★ Done only if superior breaks.

Silicone Oil : injected into vitreous cavity.

Removed after around 8-12 weeks

- Oil needs to be

complication : Hyperoleon
(or)

- Side effect : If Silicone oil leaks into AC, it gathers Superiorly

External

Scleral buckling

If Retina immobile

Proliferative vitreo Retinopathy (PVR) : Leads to formation of membrane and scarring.

Vitrectomy ✓
+
SRF drainage ✓

Lincoff rules : Rules that help to locate the break in retina. ✓

Tractional retinal detachment

00:26:54

No break.
No photopsia, no floaters.
Occurs due to static vitreoretinal traction.

Causes :

1. most common: Diabetic retinopathy

Active space

2. Retinopathy of prematurity ✓
3. Penetrating trauma ✓
4. Sickle cell retinopathy: Presents with Roth spots (hemorrhage with a clear centre).

Clinical features:

1. Loss of vision: Gradual and painless
2. No photopsia, no floaters
3. Does not extend till Ora serrata
4. Detached retina is concave in shape

Rx: vitrectomy + Tamponade

Exudative retinal detachment

00:30:58

Causes:

m/c/c: choroidal melanoma
Toxemia of pregnancy
Coat's ds
VKH Sx
CSR

C/F: LOV: Sudden painless
Convex detatched Retina
* Shifting fluid * : Hallmark

Rx cause

Ⓡ	Ⓣ
Sudden painless	Gradual Painless
Photopsia Floaters ⊕	⊖
extends upto ora serrata	⊖
Detatched Retina : Convex	Concave

Active space

Active space

CORNEA

Cornea is the transparent anterior part of the external coat of the eye covering the iris and the pupil and continuous with the sclera.

Investigations: Keratometry, Pachymetry and Corneal topography

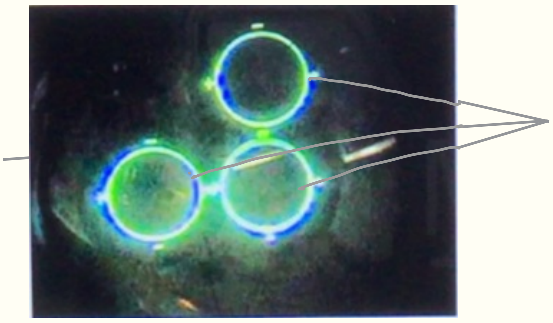
00:01:25

Keratometry :

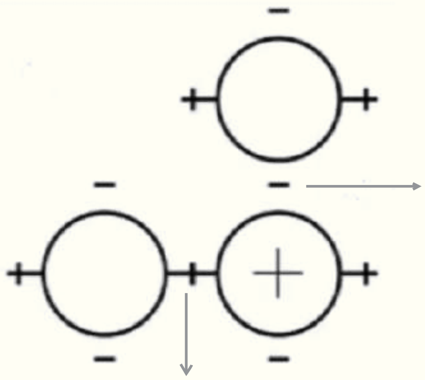
- measures the corneal curvature.
- Principle –

  Anterior surface of cornea acts as a mirror

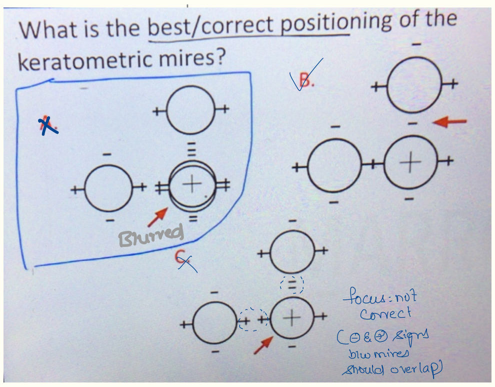
  Size of the image formed varies with curvature
- Image is seen in form of keratometric mires.


Keratometric mires

- Correct positioning of mires :


minus sign b/w vertical mires overlaps

Plus sign b/w horizontal mires overlaps


What is the best/correct positioning of the keratometric mires?
A.
B.
C.
Blurred
focus: not correct
(⊖ & ⊕ signs b/w mires should overlap)

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more curvature: vertical axis

- mires horizontally oval – with the rule astigmatism.
- mires vertically oval – against the rule astigmatism.

↳ more curvature: Hor. axis

⇒ - Pulsating mires – Keratoconus.

2. Pachymetry :
   - measures corneal thickness.
   - Normal corneal thickness – approx. 0.54mm, or 540microns. (Corneal thickness – ↑ towards periphery)

3. Corneal topography :
   - Identifies shape of corneal surface. (Ⓝ: smooth, convex)
   - Done using –
     a. Placebo disc : b/o Reflection on Cornea
     b. Orb-scan (measures corneal thickness + topograph).
     c. Penta-cam (measures corneal thickness + curvature + elevation of maps of anterior surface + elevation of maps of posterior surface + anterior segment imaging).

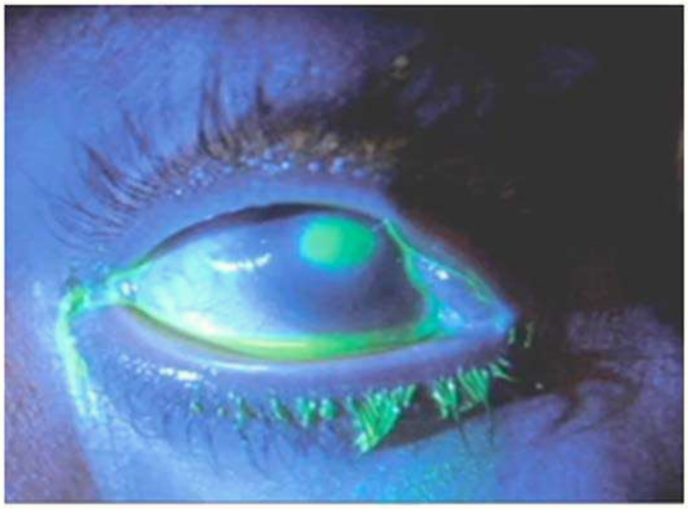
→ Placebo disc

Investigations: Corneal staining, Confocal microscopy and Specular microscopy

00:11:21

- Corneal staining :
  a. Fluorescein dye –
  - Stains area of denuded (broken) epithelium.
  - Stains floor/base of ulcer.
  - Visualized under cobalt blue light.
  - Other uses – Goldman's AT. ✓
    - For diagnosing dry eye. ✓
    - Jones's dye test (lacrimation assessment). ✓
    - Seidel's test (to detect leakage in case of corneal perforation) – dye entering Aqueous

Active space

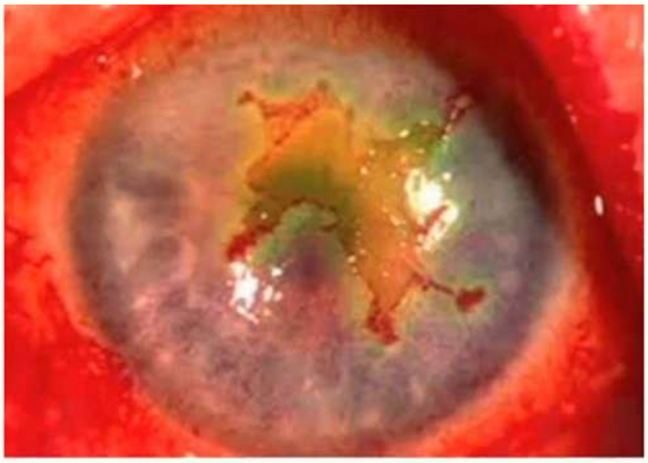


Fluorescein Staining

b. Rose Bengal dye – (Red/pink)

Stains devitalized/necrotic tissue.

Stains margin of ulcer. * (debris - collect @ margins)



Rose Bengal Staining

c. Alcian blue dye –

Stains mucin and filaments. *

d. Lissamine green – Specific

It doesn't stain the healthy ocular surface.

5. Confocal microscopy :

Non-invasive technique for in-vivo imaging of all layers of living cornea.

Active space

6. Specular microscopy :

For corneal endothelial cell count.

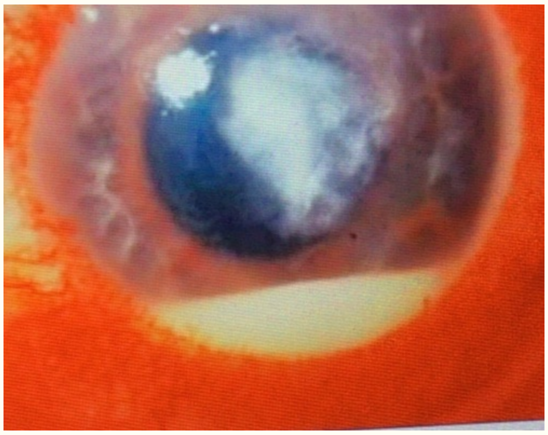
Corneal ulcer

00:20:44

Corneal ulcer is a discontinuity in the corneal epithelium associated with infiltration and necrosis of underlying layers.

Bacterial corneal ulcer:

- most common cause in world – staphylococcus Aureus.
- most common cause in India – streptococcus pneumoniae/ pneumococcus (also known as ulcus serpens or hypopyon corneal ulcer).



- most common cause of bacterial ulcer in contact lens users – pseudomonas (presents with green colour discharge).
- most common cause of bacterial ulcer in a post refractive surgery patient – atypical mycobacterium.

- Nocardia –

  Causes wreath pattern ulcer, or pin head ulcer.

  Resembles fungal corneal ulcer.

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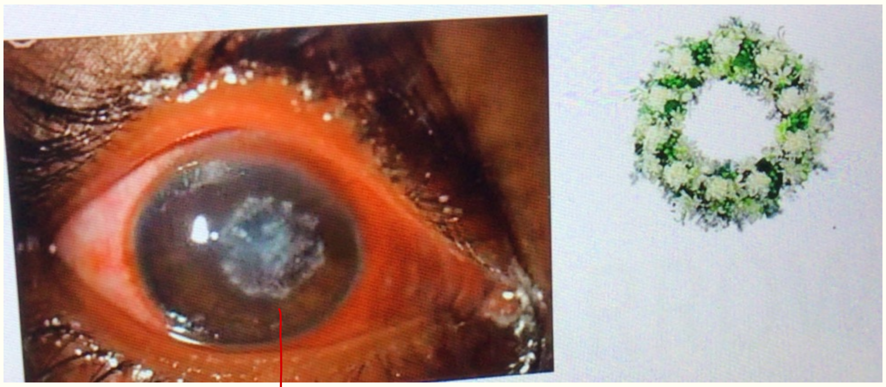


Wreath pattern ulcer

- Bacteria invading intact cornea include –
  - a. Neisseria gonorrhoea. ✓
  - b. Neisseria meningitidis. ✓
  - c. Haemophilus aegypticus.
  - d. Corynebacterium diphtheriae.
  - e. Listeria.
  - f. Shigella.

Pathogenesis of bacterial corneal ulcer

00:29:50

Initial stage : →	Progressive stage : →	Regressive stage : →	Scar/ cicatrisation
Saucer shaped ulcer with margin projecting above the ulcer surface.	Infiltration with PMNs (polymorphonuclear cells).	Floor and edges become smooth and, ==corneal vascularization occurs.==	

Clinical features bacterial corneal ulcer

00:32:11

Pt - pain, Blurring of vision
Redness
FB sensation
Lacrimation c̄ photophobia

Signs :1- Hypopion
- Sterile
- Mobile

2 Keratouveitis (toxins)

3- Ant staphyloma

4. Descematocele : Herniation of D-M

5. perforation : IOP ↓
Iris prolapse
Ant. synechiae
Ant capsular cataract
pthysis : Atrophic sunken eyeball

Mx: a send corneal scraping
→ Gram staining & culture
↓
Start Antibiotics (No emp. ℞)

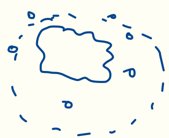
b Supporting therapy : cycloplegics - Atropine

* Topical steroids = CI (impede healing process)
+
Promote corneal thinning)

Fungal corneal ulcer

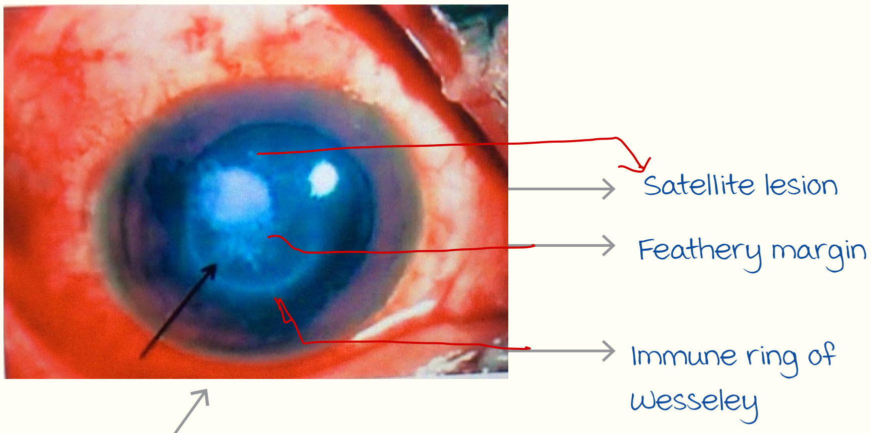
00:39:37

- most common fungi – aspergillus. (filamentous fungi c̄ septate hyphae)
- Cause –
  Patient gives h/o trauma with vegetative material. (farmer)
- Signs – out of proportion to symptoms
  Dry looking ulcer with feathery margins.
  Satellite lesions.



* Sterile immune ring of wesseley. * : around ulcer (Ag-Ab Rx$_2$)
Hypopyon (unsterile and immobile).

Active space



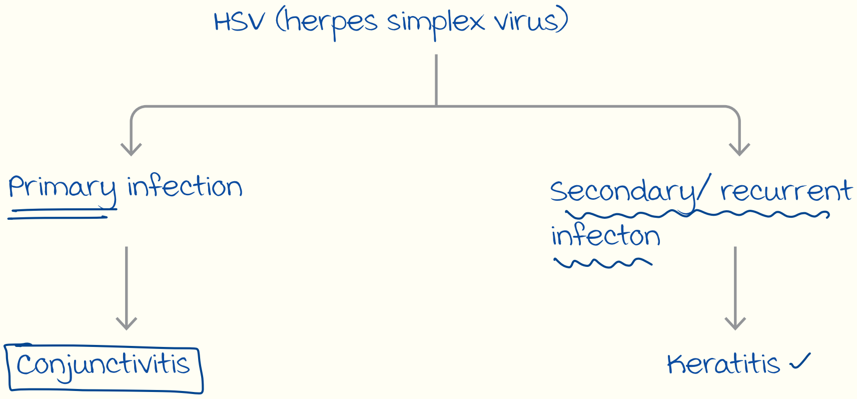
Fungal corneal ulcer

- Treatment –

  1. Drug of choice – 5% natamycin eye drops.
  2. ⇒ Supporting therapy – cycloplegics (atropine).
  3. Steroids – contraindicated.
  4. Amphotericin – B 0.15%, Fluconazole 0.3% → If candida infection.

Viral keratitis

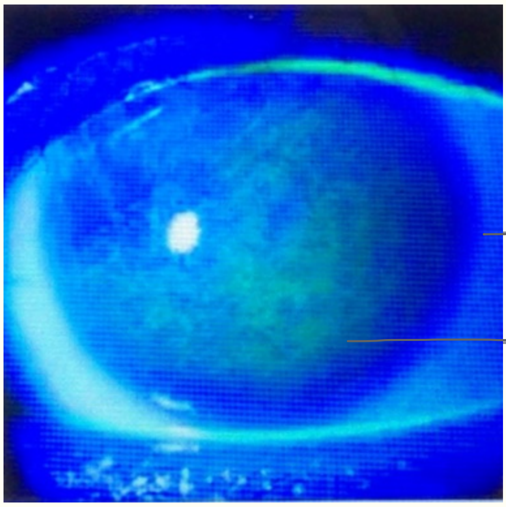
00:46:03



- Clinical features of HSV keratitis :
  1. Epithelial lesion –
     a. Due to active viral infection.
     b. Superficial punctate keratitis.
     c. Dendritic ulcer.
     d. Geographic ulcer.

Active space

Telegram--@latestpgnotes

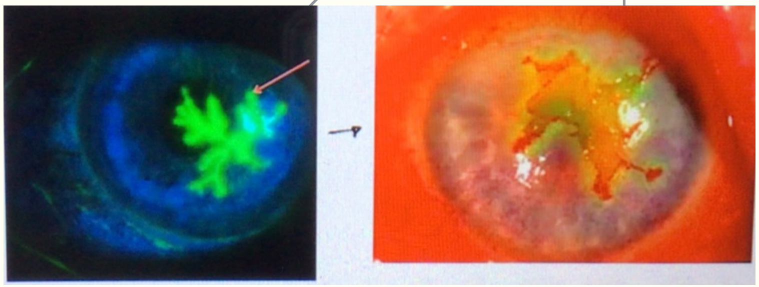


Stained with fluroscein dye

Punctate lesions (dot like lesions)

Branched pattern

map like lesion



Dendritic Ulcer

Geographic Ulcer

Treatment of epithelial lesions –

e. 3% acyclovir eye ointment – 5 times a day for 2 weeks.

f. Steroids – contraindicated.

2. Stromal lesion –

a. Necrotising keratitis.

⇒ b. Immune stromal keratitis (type 3 hypersensitivity rection).

Treatment of stromal lesion –

a. Acyclovir ointment.

b. Steroids. – can be given since the lesion is stromal

3. Endothelial lesion –

a. Disciform keratitis (type IV hypersensitivity rection).

☆ b. Diffuse endothelitis.

Treatment of endothelial lesion –

a. Disciform keratitis – acyclovir ointment + steroids.

b. Diffuse endothelitis – topical steroids. ✗

Topical Steroids

CI : Epithelial lesions of HSV
Bac & fungal Corneal ulcer.

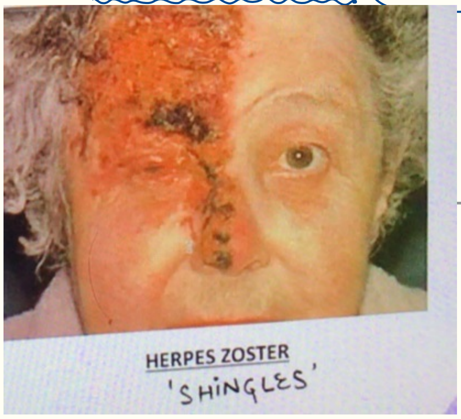
4. Neurotrophic ulcer or metaherpetic ulcer –

a. Non-infective lesion.

b. Occurs due to corneal anaesthesia.
↳ Hallmark : HSV

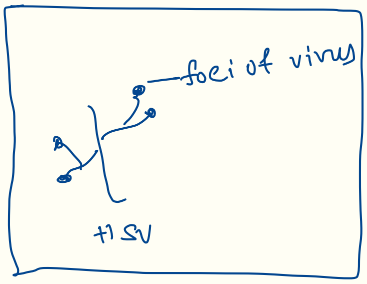
Herpes zoster ophthalmicus (HZO) 00:55:20

- Caused by HZV (Herpes zoster virus).
- most common in 6th – 7th decade in females.

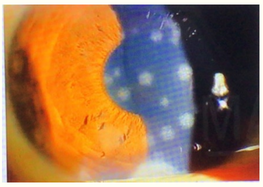


→ Herpes zoster

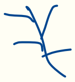
Hutchinson's sign – pustule at tip of nose has greater correlation with ophthalmic manifestation.



- Clinical features of HZO –



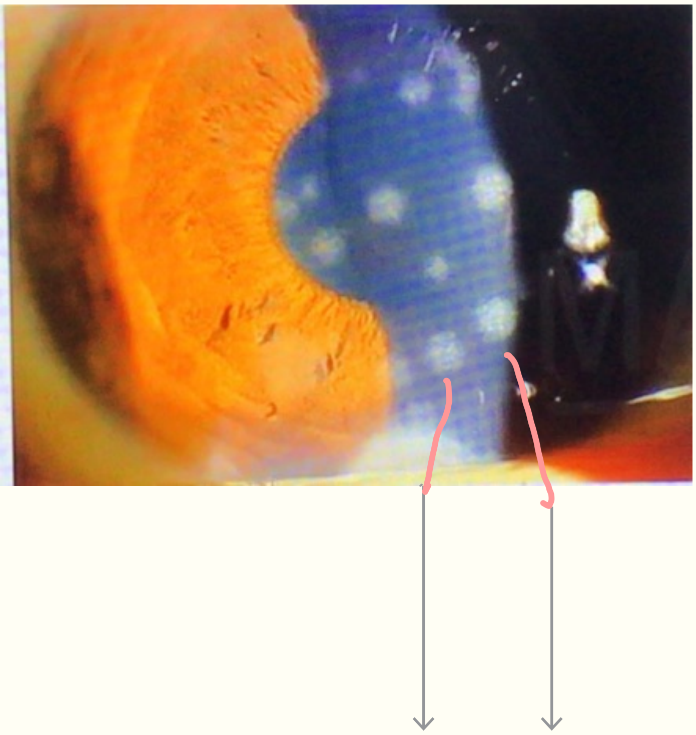
1. Pseudo dendritic ulcer.



No Terminal buds

2. Nummular keratitis – coin shaped subepithelial ✱ lesions surrounded by stromal haze.
3. Mucous plaque keratitis.
4. Sclerokeratitis – least common.✓ ~~★★~~
5. Neurological – IIIrd CN palsy ✗
6. Episcleritis.

Rx : oral Acyclovir 800mg × 5/day × 2w



Coin shaped lesions

- Treatment of HZO –
  Oral acyclovir 800mg, 5 times a day for
  2 weeks.

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

Acanthamoeba keratitis and Interstitial keratitis

01:00:19

- most commonly occurs in contact lens users (using tap water, contaminated solutions), Swimming pool wearing lens
- Severe pain >>>> signs.
- Signs –
  Pseudo dendrites. (≡ HZV)
  Ring abscess.
  Radial Keratoneuritis.

- Culture for media – non nutrient agar enriched wit E. coli. *

- Treatment –
  1. Drug of choice – PHMB (polyhexamethylene biguanide) 0.02%.
  2. Propamidine. ✓
  3. Chlorhexidine.

Interstitial Keratitis

- Only stromal inflammation.
- most commonly caused by congenital syphilis.

Keratoconus

1:05:14

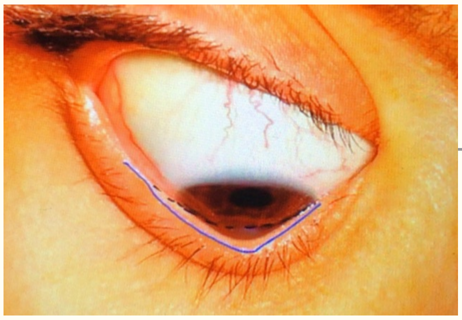
- Keratoconus is a form of corneal ectasia.
- It is non inflammatory, progressive and usually bilateral.
- Clinical features of Keratoconus –

1. Hallmark – stromal thinning (paracentral) with apical protrusion (usually inferior)

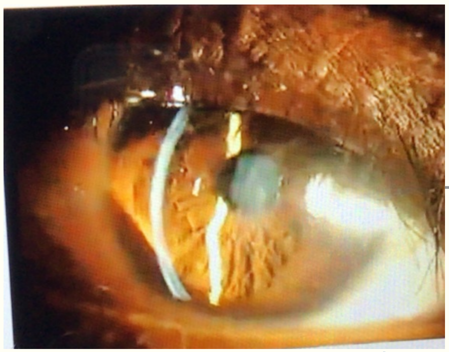
↑Curvature

Apex

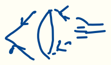
2. myopia + irregular astigmatism. ✓
3. munson's sign – bulging of lower eyelid on downgaze.
4. Rizzutis sign – arrow head pattern of light, formed over nasal limbus when light is projected temporally.



munson's sign :
Bulged lower eyelid
on down gaze



Rizzutis sign:
Arrow head pattern
of light over nasal
limbus
(light refracted by
the cone)



Active space

5. On retinoscopy – scissoring reflex.*
6. On ophthalmoscopy – oil droplet reflex.
7. Fleisher's ring – deposition of iron/hemosiderin.
8. Acute hydrops – due to influx of aqueous into cornea in Descemet's membrane rupture.

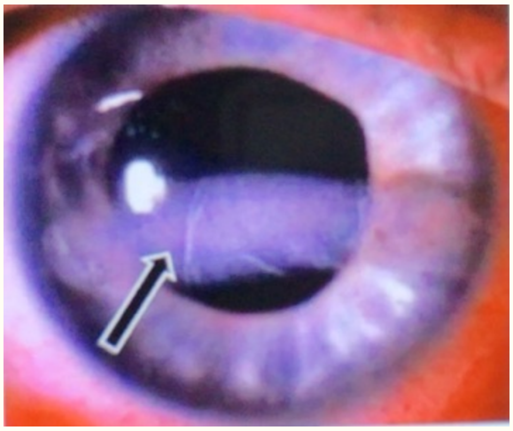
Investigation: Corneal Topography
↓
Symmetric bow tie appearance

Rx: 1. Rigid contact lens
2. Keratoplasty
3. C3R (Corneal Collagen crosslinking using B2 (Riboflavin))

Anesthise the eye
↓
Central 8-10mm corneal epithelium = removed
↓
0.1% Riboflavin solution put on eye
↓ 30 min
Expose cornea to UVA (370nm) light

Band shaped keratopathy and Arcus senilis

01:17:18

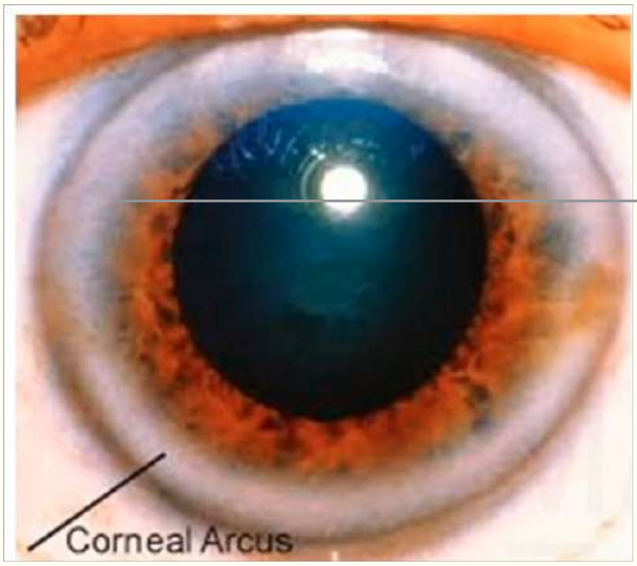


Band Shaped Keratopathy

- Deposits of calcium in bowman's layer and stroma (superficial).
- Cause in adults – uveitis (chr.) ☆
- Cause in children – Still's disease.
- Treatment – chelation with EDTA.

Arcus senilis

- Occur in old age. , paralimbal lipid dep.
- Involves stroma.
- Clear interval of VOGT (clear space b/w arcus and limbus).



Clear space b/w arcus and limbus

Keratoplasty

01:22:09

- Commonly known as corneal transplantation

- Keratoplasty
  - Penetrating keratoplasty
    - Full thickness
  - Lamellar keratoplasty
    - Partial thickness
      - DALK (deep anterior lamellar keratoplasty)
      - DESK (Descemet's stripping endothelial keratoplasty)

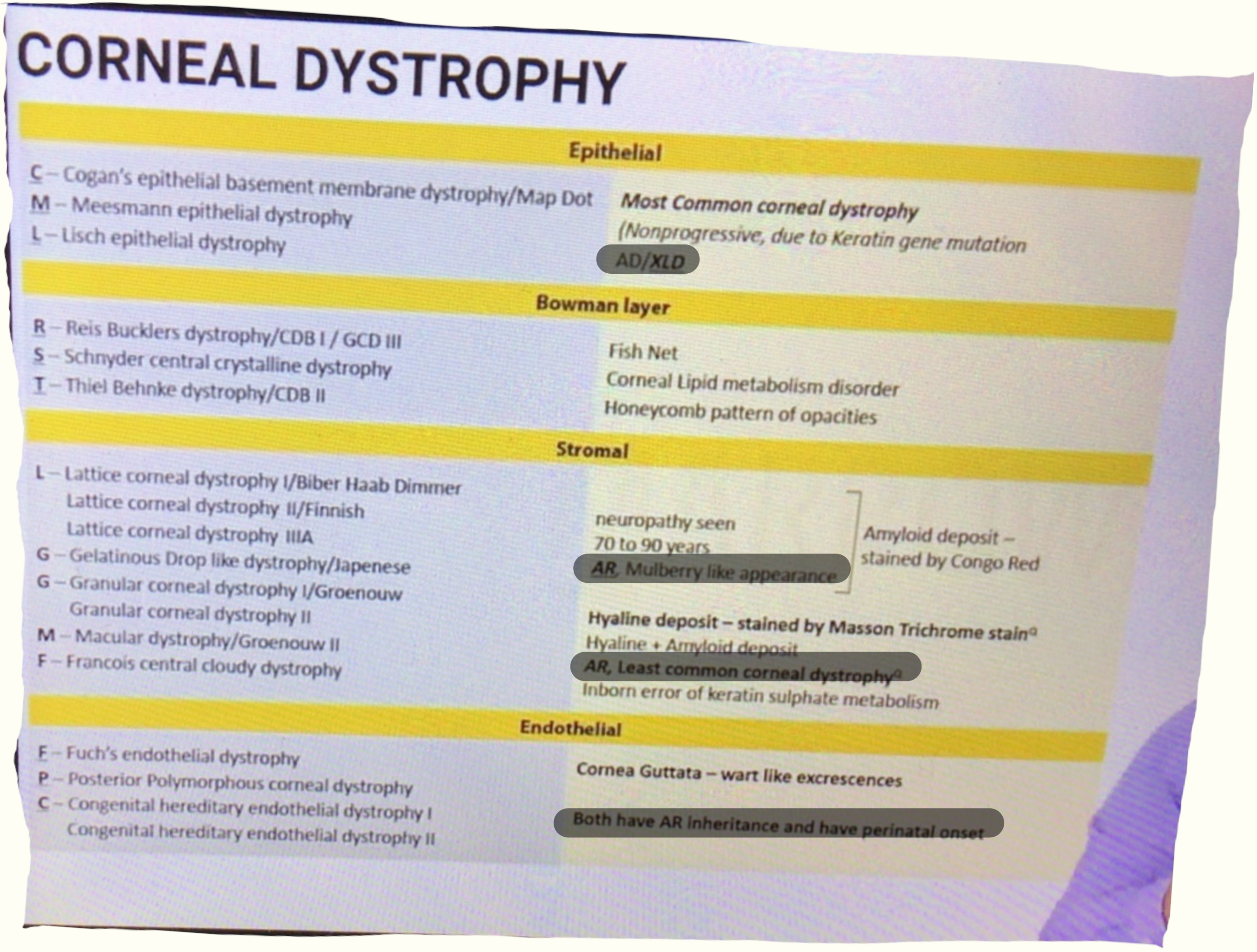
- DALK –
  Corneal tissue is removed up to Descemet's membrane.
  Indicated in keratoconus with acute hydrops.
- DESK –
  Removal of only endothelium and Descemet's membrane.
  Indicated in pseudophakic bullous keratopathy.
  ↳ bulla in cornea
- For corneal transplantation normal donor graft size should be 0.25mm larger than host opening.

↓ To extract cornea (only central part = extracted)

- Storage media for corneal transplantation-
  1. Short term (<48 hours) – 4 degree Celsius ; moist chamber.
  2. Intermediate (upto 2weeks) – Mc Karry Kauffman media.
  3. Long term (upto 35days) – cryopreservation (-70 degree Celsius).
- Graft rejection –
  a. most common rejection – endothelial. *
  b. Clinical features – Khodadoust line.
  - Linear keratic precipitates.

Corneal dystrophy

01:29:11



CORNEAL DYSTROPHY

Epithelial	
C – Cogan's epithelial basement membrane dystrophy/Map Dot	***Most Common corneal dystrophy***
M – Meesmann epithelial dystrophy	*(Nonprogressive, due to Keratin gene mutation*
L – Lisch epithelial dystrophy	AD/*XLD*
Bowman layer	
R – Reis Bucklers dystrophy/CDB I / GCD III	Fish Net
S – Schnyder central crystalline dystrophy	Corneal Lipid metabolism disorder
T – Thiel Behnke dystrophy/CDB II	Honeycomb pattern of opacities
Stromal	
L – Lattice corneal dystrophy I/Biber Haab Dimmer	Amyloid deposit – stained by Congo Red
Lattice corneal dystrophy II/Finnish	neuropathy seen
Lattice corneal dystrophy IIIA	70 to 90 years
G – Gelatinous Drop like dystrophy/Japenese	AR, Mulberry like appearance
G – Granular corneal dystrophy I/Groenouw	**Hyaline deposit – stained by Masson Trichrome stain^{a}**
Granular corneal dystrophy II	Hyaline + Amyloid deposit
M – Macular dystrophy/Groenouw II	***AR, Least common corneal dystrophy^{a}***
F – Francois central cloudy dystrophy	Inborn error of keratin sulphate metabolism
Endothelial	
F – Fuch's endothelial dystrophy	**Cornea Guttata – wart like excrescences**
P – Posterior Polymorphous corneal dystrophy	
C – Congenital hereditary endothelial dystrophy I	**Both have AR inheritance and have perinatal onset**
Congenital hereditary endothelial dystrophy II	

Telegram--@latestpgnotes

CONJUNCTIVA

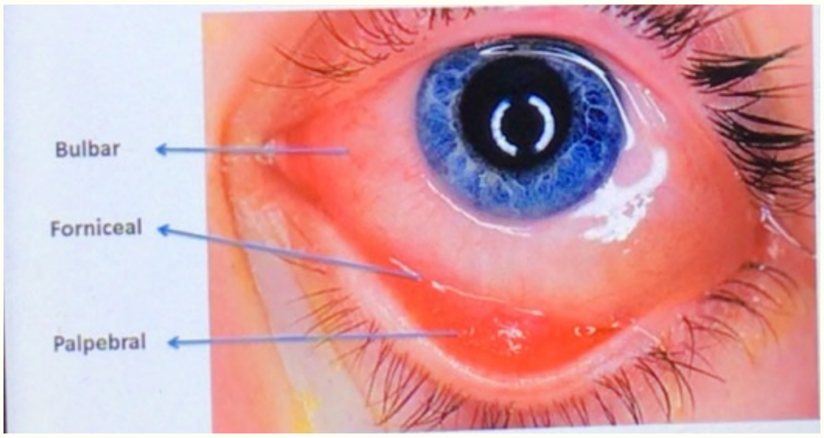
Parts of conjunctiva

00:00:20

Conjunctiva is transparent/translucent mucous membrane.
There is no conjunctiva over the cornea.

Three parts :

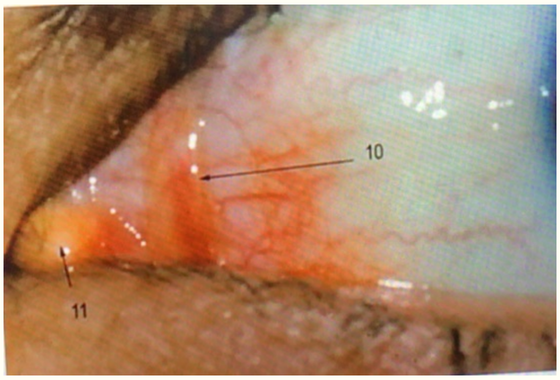
1. Bulbar conjunctiva - covers the sclera.
2. Palpebral conjunctiva - lines the inner surface of eyelids.
3. Forniceal conjunctiva- shallow depression between palpebral and bulbar conjunctiva.



There are two specialized folds :

10) • Plica semilunaris - represents the nictitating membranes.

11) • Caruncle - contains hair follicles.



10. Plica semilunaris
11. Caruncle

Active space

Histology of conjunctiva

1. Outermost layer –
   Epithelium (nonkeratinised stratified squamous) (≡ Cornea)
   It contains – 1. Goblet cells, which secrete mucin on parasympa thetic stimulation (mucin forms the innermost layer of tear film).
   2. melanocytes
   3. Langerhans cells

2. Adenoid/lymphatic layer – develops after 3 months of age (ie No lymphoid Rx2 <3y Age)
3. Fibrous layer

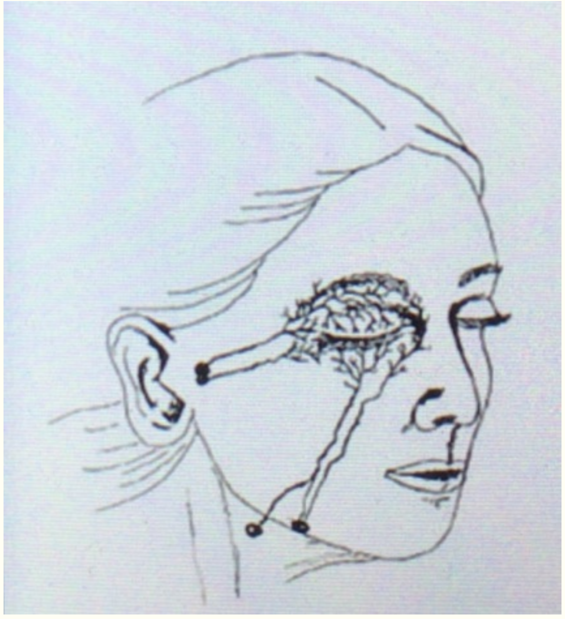
Lymphatic drainage of conjunctiva

00:06:09

The lymphatic drainage in the eye occurs only from the conjunctiva.

It can drain

- medially
  - Submandibular lymph nodes
- laterally
  - preauricular & superficial parotid lymph nodes



* Lymph flows as tear flow

Active space

Telegram--@latestpgnotes

Conjunctivitis

00:09:07

Eye flu Symptoms :

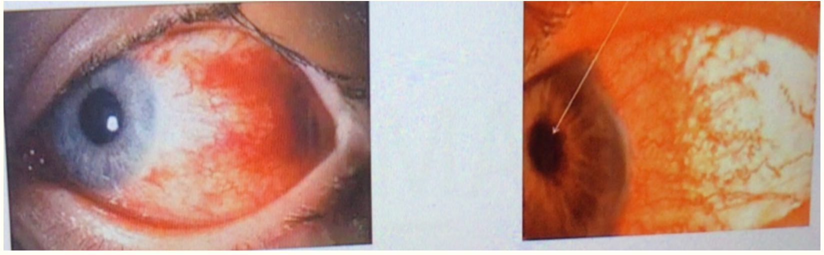
Redness, stickiness, foreign body sensation, lacrimation, itching (allergic). * If pain and loss of vision present, there is cornea involvement. *

signs :

1. Congestion/hyperaemia :

Redness

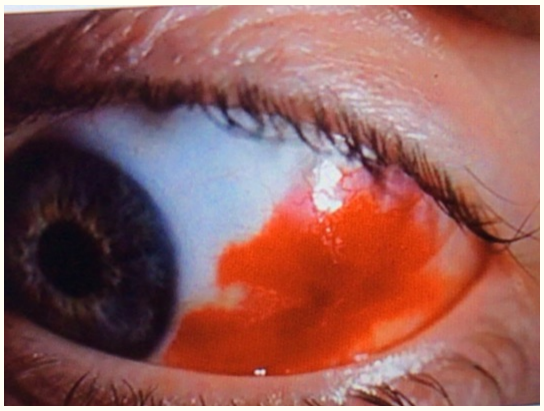
- Conjunctival
  - Seen in conjunctivitis
- circumcorneal
  - seen in glaucoma, uveitis, corneal ulcer



2. Subconjunctival haemorrhage

No treatment required.

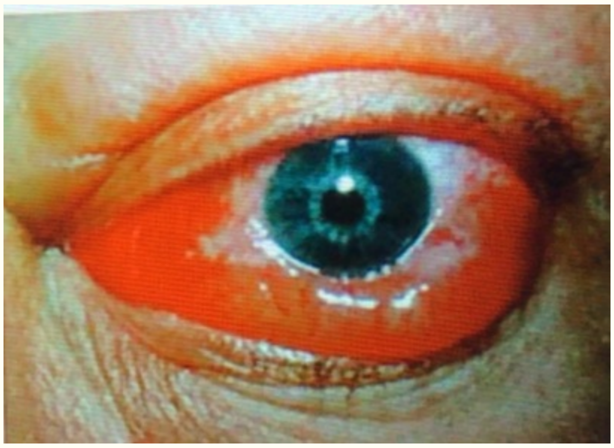
Resolves spontaneously in 2 weeks. *



Hemorrhage

3. Chemosis

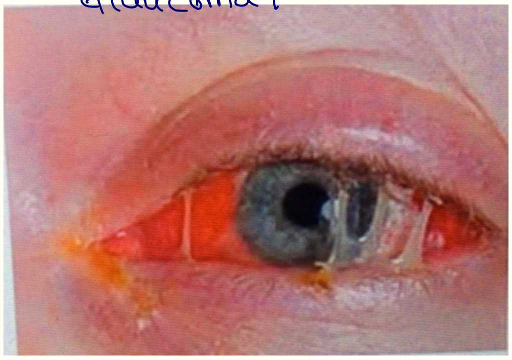
It is swelling of the bulbar conjunctiva.



Discharge

It can be mucopurulent or watery. It leads to stickiness and coloured halos.

MPC → washed
Cataract
Glaucoma
fincham



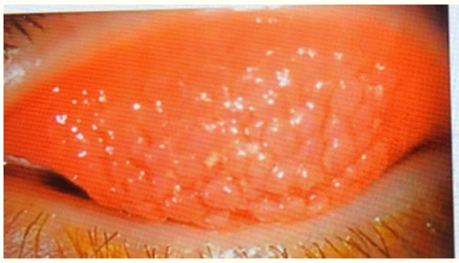
Discharge:
Sticking together of eyelids in the morning
Colored halos

Inflammatory reactions of conjunctiva

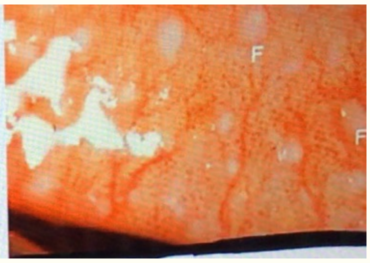
00:14:31

Two types
- Papillae
  - Hypertrophied vessel
- Follicles
  - lymphoid aggregates

i.e not seen in 1st 3months



Papillae



Follicles

Discharge	Papillary reaction	Follicular reaction
mucopurulent	Bacterial conjunctivitis	Chlamydia conjunctivitis
watery	Allergic conjunctivitis	Viral conjunctivitis

Bacterial conjunctivitis :papilla + mucopurulent d/c

00:18:19

1. Acute Mucopurulent :

m/c/c : S. Aureus

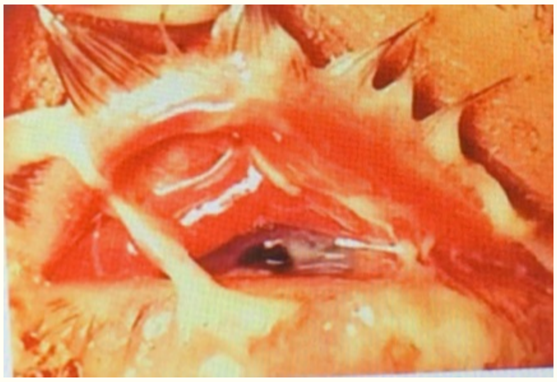
Rx : Topical Abs

2. Acute Purulent / Hyperacute Conj / Blenorrhoea :

m/c/c : Neisseria Gonorrhoea

↓

Oculogenital Tx



a. Copious d/c

b. Swelling of eyelids (overhanging)

c. Intense pain

d. Pre-auricular LN' pathy

Rx : IM Ceftriaxone 1g single dose

↓ f/b

Oral Erythromycin x 2W

Active space

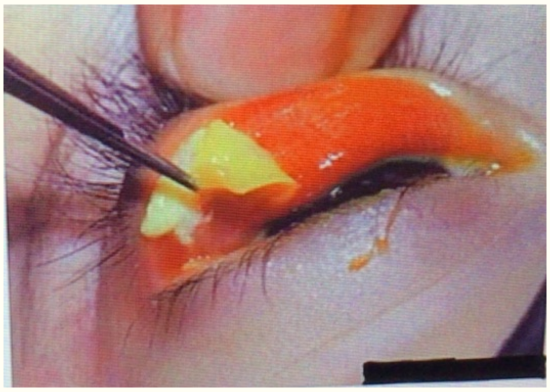
3- Acute membranous conjunctivitis

MCC in non immunized children - C. diphtheria
MCC in adults/overall- pneumococcus ✱
The membrane is fused with the underlying epithelium and it bleeds on removal.

Bleeds on removal
Firmly adherent

Acute pseudomembranous conjunctivitis

The membrane does not fuse with underlying epithelium and does not bleed on removal.

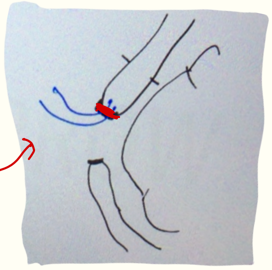


Peeling no bleeding

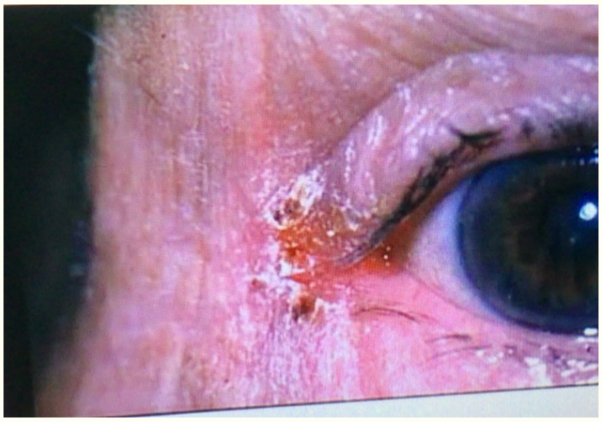
Active space

Angular conjunctivitis/ diplo bacillary ✱

MCC moraxella lacunata/axenfeld
C/F - excoriation of skin at medial canthi
Reddening of intermarginal strip
Blepharitis



Treatment – Tetracycline 1% eye ointment for 2 weeks + Zinc boric eye drops



Viral conjunctivitis

00:28:38

Adenoviral conjunctivitis

Two types of manifestations :

- EKC (epidemic keratoconjunctivitis) – serovar 3,4,7
- PCF (pharyngoconjunctival fever) – serovar 8,19, 37, associated with preauricular lymphadenopathy

Apollo conjunctivitis/ acute haemorrhagic conjunctivitis

Causes are : NEET 18

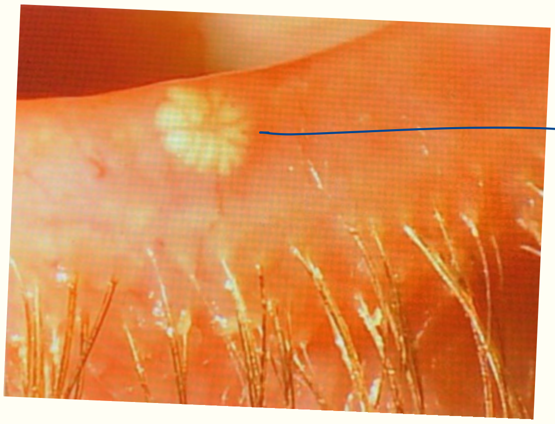
1. Picornavirus – enterovirus type 70, coxsackie A24
2. Adenovirus type 11

PACE

molluscum contagiosum

C/F – unilateral, painless node with umbilicated appearance.

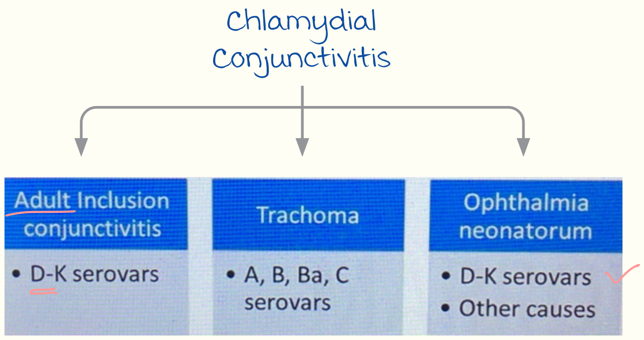
m



sheds virus

Active space

Chlamydia conjunctivitis 00:33:55



Chlamydial Conjunctivitis

Adult Inclusion conjunctivitis	Trachoma	Ophthalmia neonatorum
• D-K serovars	• A, B, Ba, C serovars	• D-K serovars ✓ • Other causes

Trachoma

It is also known as Egyptian ophthalmia.

It is caused by chlamydia trachomatis AB,Ba,C serovars.

* Transmission – fingers, flies, fomites. *

mc in children < 10years of age.

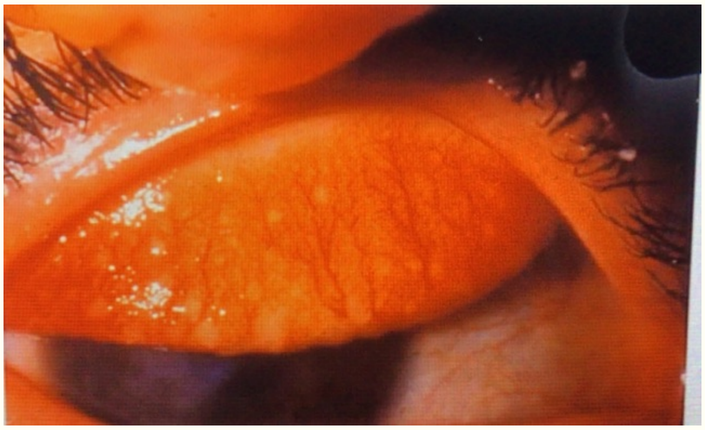
It is type IV hypersensitivity reaction and active inflammation with cicatrization present together.

Clinical signs of trachoma 00:36:50

1. Sago grain follicles

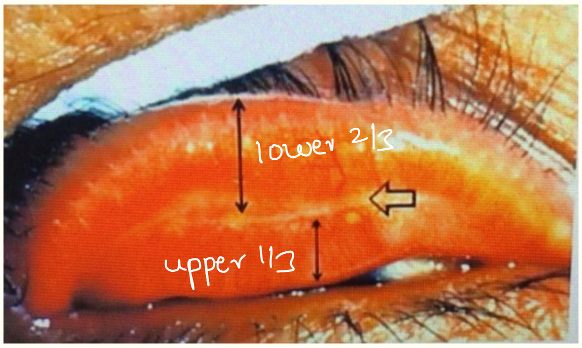
   mc seen in upper palpebral conjunctiva

   These follicles show necrosis and leber cells on HPE



2. Cicatricial sign

   Arlt's line – line of cicatrization present between the lower 2/3rd and upper1/3rd.

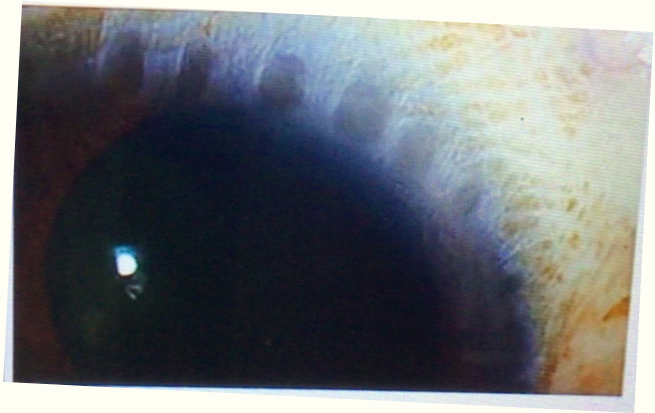


Arlt's Line

3. Herbert pits

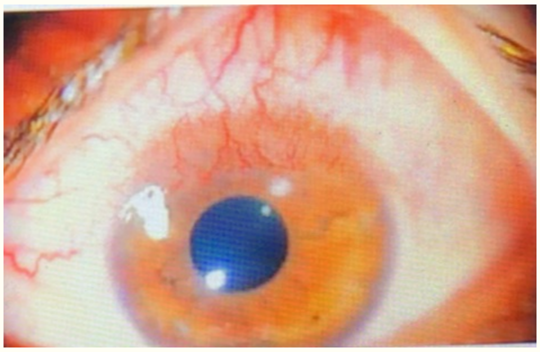
   It is pathognomonic for trachoma

   It is due to healing of limbal follicles



4. Pannus

   It is vascularisation of cornea superiorly.



Pannus

Telegram--@latestpgnotes

Only complication – corneal ulcer (due to trichiasis) → inward growth of eyelashes d/t cicatrisation

↓

Heals to form scar

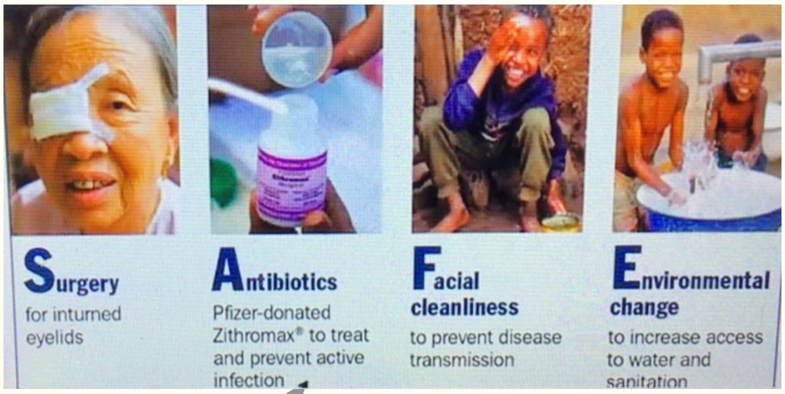
↓

Leads to loss of vision

Treatment and classification of trachoma

00:43:50

SAFE strategy : WHO : 1996


Surgery
for inturned eyelids
Antibiotics
Pfizer-donated Zithromax® to treat and prevent active infection
Facial cleanliness
to prevent disease transmission
Environmental change
to increase access to water and sanitation

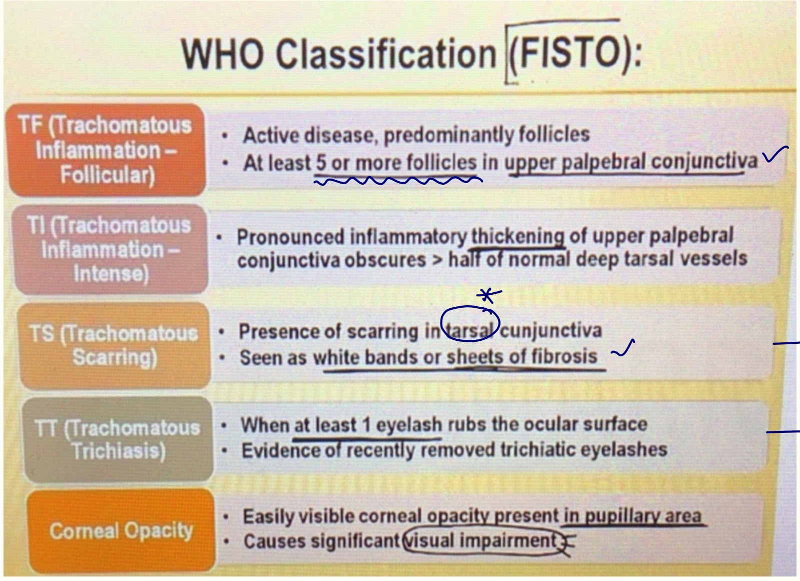
DOC : Azithromycin 1 gm

Tx :

- If prevalence >10% in children aged 1-9y → Mass prophylaxis
- 5-10% : Rx Affected children
- <5% : only F, E

Active space

Classification of trachoma



WHO Classification (FISTO):

TF (Trachomatous Inflammation – Follicular)	• Active disease, predominantly follicles • At least 5 or more follicles in upper palpebral conjunctiva ✓
TI (Trachomatous Inflammation – Intense)	• Pronounced inflammatory thickening of upper palpebral conjunctiva obscures > half of normal deep tarsal vessels
TS (Trachomatous Scarring)	• Presence of scarring in tarsal* cunjunctiva • Seen as white bands or sheets of fibrosis ✓
TT (Trachomatous Trichiasis)	• When at least 1 eyelash rubs the ocular surface • Evidence of recently removed trichiatic eyelashes
Corneal Opacity	• Easily visible corneal opacity present in pupillary area • Causes significant visual impairment

→ Rx by Ab

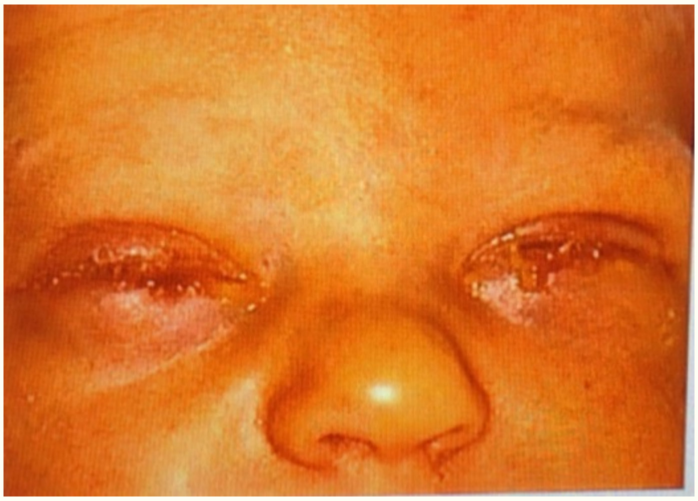
— Inactive : No Rx

→ Sx

Ophthalmia neonatorum

00:48:05

It is conjunctivitis in neonates (<28 days of onset).



Onset	Organism
1) c̄in 6hrs	Chem. Conjunctivitis (AgNO3)
2) 24-48h	N. Gonorrhoea (most severe)
3. 2-5d	other bacteria
4. 5-7d	HSV-2
5- >1week	C. Trachomatis (D-K) –m/c

Prevention :

1. Crede's method – topical 1% silver nitrate (not recommended anymore).
2. 0.5% erythromycin or 1% tetracycline– given within 1 hour of birth.

Telegram--@latestpgnotes

Allergic conjunctivitis

00:52:20

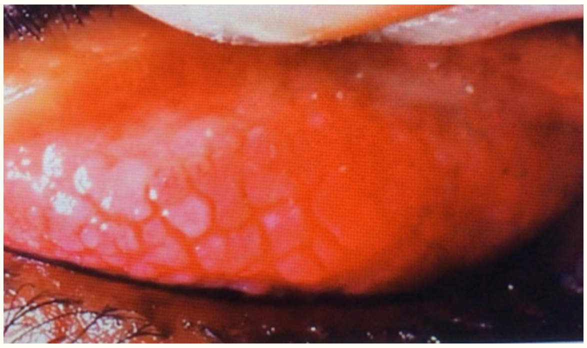
It presents with a papillae reaction with watery discharge and itching.

Vernal keratoconjunctivitis/spring catarrh

- It is type I hypersensitivity reaction (e.g., pollen).
- MC in boys (5–15 years).
- MC in spring and summers.
- History of atopy present.

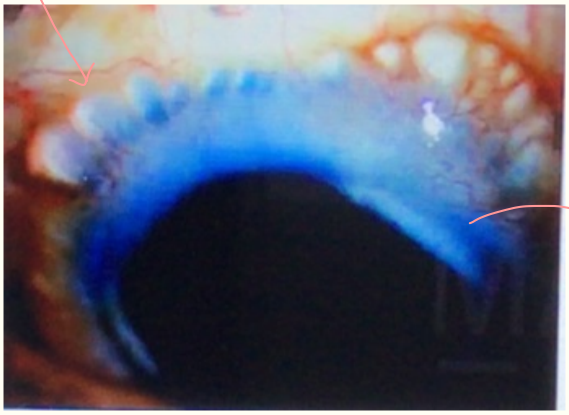
Clinical signs

1. Papillary hypertrophy – cobblestone appearance



2. Horner trantas dots

Allergy
↑
It is a collection of eosinophils & epithelial debris on the limbus



old age
↑
3. Pseudo gerontoxon

It is a paralimbal grey white band of lipid deposition seen in children.

Active space

⇒ Old Age :Arcus senilis
↓
Paralimbal Grey white lipid deposition

4. Shield ulcer ✓
5. Dennie morgan line- an extra lower lid crease
6. maxwell lyon sign- ropy discharge.

★ The excessive itching - leads to greater incidence of Keratoconus *

Treatment of Vernal keratoconjunctivitis 01:01:10

✓ 1. Cool compresses
✓ 2. Topical antihistamines
✓ 3. Topical mast cell stabilizers
4. DOC olopatadine or alcaftadine
⇒ 5. Topical steroids in acute exacerbations * ★

Atopic Keratoconjunctivitis

It occurs in old age 20-50 years.

Eyelids - hertoghe's sign the lateral eyelashes are lost. ★

Giant papillary conjunctivitis

It is >1mm in diameter.

Type IV hypersensitivity reaction.

It is mechanically induced by - contact lenses ✓
Ocular prosthesis ✓
Protruding sutures. ✓

Treatment - Antihistamines.

Phlyctenular keratoconjunctivitis 01:06:27

It is type IV hypersensitivity reaction. — in response to endogenous allergens

mcc in India - tuberculosis.

mcc in western countries - staphylococcus aureus.

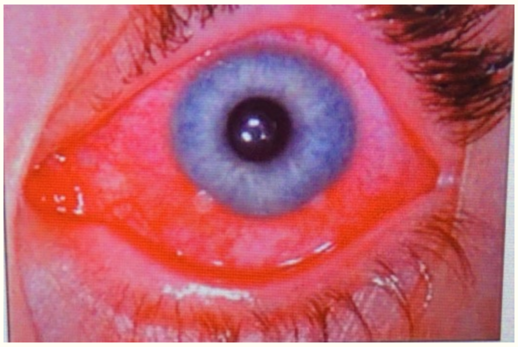
Clinical sign :

Phlycten - it is a nodule near the limbus, grows towards the cornea and ulcerates, giving rise to three types of ulcer :

- Sacrofulous ulcer
- Fascicular ulcer
- miliary ulcer

Active space

Treatment – steroids.



Pterygium

Nasal - common

01:10:25

A triangular, fibrovascular growth of degenerative subconjunctival tissue over the cornea, destroying bowman's and superficial stroma is called pterygium.✓

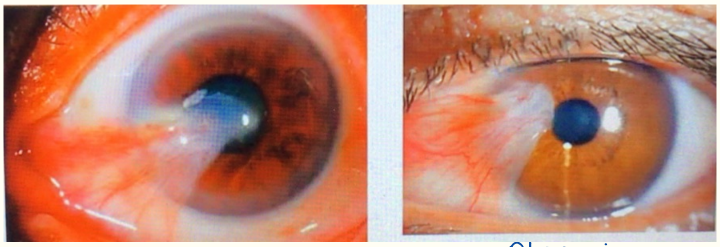
It is associated with UV ray exposure.*

It is associated with dust, humidity.✓

It occurs also due to limbal stem cell deficiency. → ⊕ MMP ↓ d/s

Clinical signs :

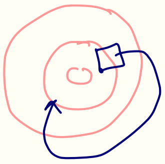
1. Triangular growth with apex towards cornea.
2. Stocker's line – deposition of iron in front of apex of pterygium.
3. Loss of vision – due to : corneal astigmatism.
   Growth encroaching on the visual axis(covers the pupillary area).



Pterygium

Treatment

Surgical excision – it has a high rate of recurrence. *



To prevent recurrence :

- Topical mitomycin C
- Autografting – graft harvested from the same eye (usually superiorly).
  Sutures or fibrin glue or autologous serum are used to fix it.

Active space

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UVEA

Anterior Uveitis

00:00:58

It is also known as Irido-cyclitis.

Involves inflammation of Iris and Pars plicata.

Causes :

1. Granulomatous Uveitis
   - a. TB
   - b. Sarcoidosis
     - It causes anterior uveitis as : Lofgren syndrome (Erythema nodosum + Bilateral hilar lymphadenopathy + Anterior uveitis).
       Heerfordt syndrome (Uveitis + Parotitis + Fever).
   - c. Leprosy
     - most common LL (Lepromatous)
     - Specific clinical feature : Iris pearls ✱
   - d. Syphilis
     - most commonly occurs in late congenital case. ✱
     - Specific clinical feature : Iris roseolas ✱
     - Poor response to steroids.
2. Non-granulomatous Uveitis
   - a. Acute
     - (1) most common cause : Idiopathic
     - (2) HLAB27 associated diseases :
       - ✓ Ankylosing spondylitis : Lower back pain + Unilateral anterior uveitis.
       - Reiter's disease : History of travel and infection with shigella ☆
         Urethritis + Arthritis + Conjunctivitis + Keratoderma blennorhagicum.
       - Psoriatic arthritis : Sausage digits + Bilateral anterior uveitis.
     - (3) Behcet's disease
       - Associated with HLA B5, B51
       - Bilateral recurrent anterior uveitis + Cold mobile hypopyon. ☆
         Non infective

(4) Inflammatory bowel disease

- more common in Ulcerative colitis. ✱

Causes of Chronic Non-granulomatous uveitis

00:10:35

b. Chronic

1. Juvenile Idiopathic Arthritis (JIA)
   - most common cause of anterior uveitis in paediatric age group.
   - Arthritis≥ 6 weeks before 16 years of age.
   - 3 types :
     - Pauciarticular
       - ≤ 4 joints involved
         - Type 1
           - Girls
           - RF negative (seronegative)
           - ANA positive
           - ☆ most commonly associated with Anterior uveitis
         - Type 2
           - Boys ✱
     - Polyarticular
       - ≥ 4 joints involved
     - stills disease ✱
       - Systemic manifestations

2. Fuch's HeterochromicIridocyclitis (FHI)
   - Clinical features :

     mnemonic : No F U C H S

     No posterior synechiae. ✓

     Floaters : Only symptom ☆

     ✱ Unilateral

     Cataract (>80% cases) : Posterior subcapsular ✱

     Heterochromia iridis : Hypochromia in the affected eye

     ✱ (Iris stromal atrophy) ✱

     ✱ Stellate Keratic precipitates ✱

specific ☆ ☆

- Amsler's sign : On gonioscopy, vessels seen in the angle bleed on paracentesis. ✱

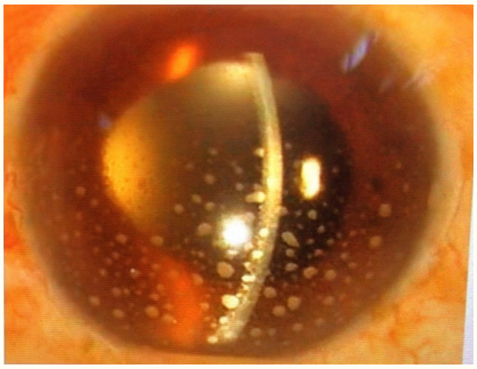
Symptoms and Signs of Anterior Uveitis

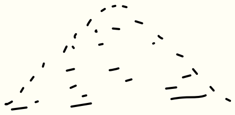
00:19:38

- Pain, Redness, Blurring of vision
- Photophobia, Lacrimation
  - ↳ Reflex
    - A – V
    - E – VII : orbicularis oculi

Signs :

1. Congestion / Hyperemia
2. Keratic precipitates : deposit on Endothelium
   - Collection of Infl. cells
   - m/c : Inferiorly [Arlt's area]





- 4 types :

1. Mutton fat KPs : Granulomatous – Macrophages
2. Fine / Granular KPs : Non-Granulomatous – Lymphocytes
3. Pigmented KPs : Chr - Non Granulomatous (brown)
4. Stellate KPs : Diffuse distribution
   - Herpetic uveitis – FHI
   - Toxoplasmosis

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

3. Aqueous cells :
   - Usually neutrophils *
   - They are markers of disease activity * : Count cells : 6–15 : 1⊕ ; 16–25 : 2⊕ ; ⋮ ⋮
4. Aqueous Flare :
   - Turbidity / translucency of aqueous due to leakage of proteins.
   - Visible due to Tyndall effect / Brownian movement.

Active space

Telegram--@latestpgnotes

Signs of Anterior Uveitis: Hyphema, Hypopyon, Iris 00:32:06

5. Hyphema :
   - Causes of Hyphema associated with anterior uveitis :

     Mnemonic : H O T S

     Herpetic uveitis

     Ophthalmia nodosa - Intra ocular caterpillar hair → uveitis

     Trauma

     Syphilis : Roseolas

6. Hypopyon :
   - Causes of Hypopyon associated with anterior uveitis :

     a. Behcet's disease : B5, B51

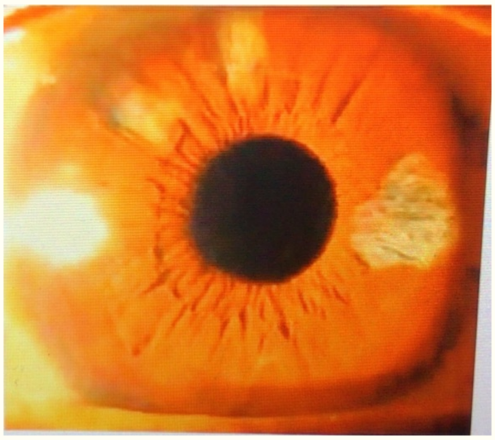
     b. HLA B27 associated disease

7. Iris

   (1) Acute cases : muddy appearance (edema)

   (2) Chronic cases - Iris atrophy : a. Sectoral : Herpetic uveitis

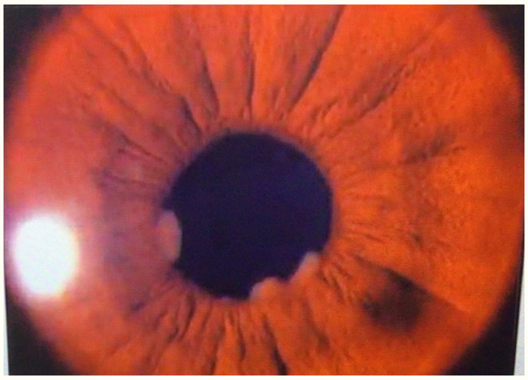
   b. Diffuse : FHI : Heterochromia (hypochromia)



Sectoral iris atrophy

   (3) Iris nodules :

   a. Koeppe nodules : Pupillary margin → Granulomatous and Non - granulomatous uveitis



Active space

b. Busacca nodules : mid iris (collarette) → Granulomatous uveitis ✱

c. Berlin nodules : Angle of Anterior chamber

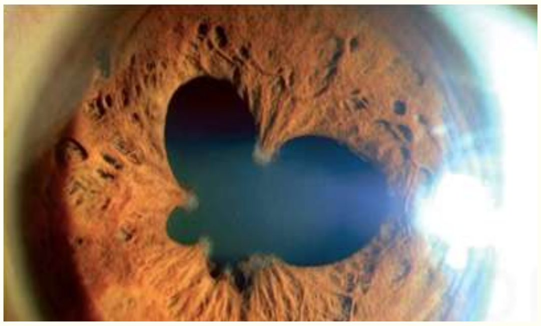
Signs of Anterior Uveitis : Pupil

00:38:58

8. Pupil

① Acute cases : miosis (spasm of spinchterpupillae m/s

(2) Chronic cases : Festooned pupil (irregularly dilated pupil formed due to incomplete posterior synechiae).



(3) Recurrent cases :

- Annular synechae or Ring synechiae (Synechiae over 360°) — only pupil

↓

Seclusio pupillae (Pupil completely in adhesion with lens : Fixed)

↓

Aqueous not able to flow from Posterior chamber to Anterior chamber.

↓

Secondary angle closure glaucoma.

(4) Total posterior synechiae : Complete Iris

- Due to exudates pouring in the Posterior chamber.

↓

Adhesion of complete posterior surface of iris with lens.

(5) Cyclitic membrane :

- Due to exudates pouring behind the lens which cover the ciliary processes and posterior surface of the lens.

↓

No aqueous secretion.

↓

Decrease in IOP known as Hypotony. ✩

(6) Occlusio pupillae

Active space

- Exudates get organised across the pupil, blocking the pupil.

Signs of Anterior Uveitis : IOP 00:49:25

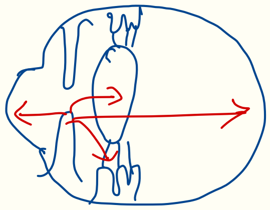
(7). IOP

1. Acute cases :
   - Raised IOP due to Trabeculitis and clogging of Trabecular meshwork by inflammatory cells : Secondary open angle glaucoma.
   - → Drug of choice : Alpha agonists (Dipvefrine) , Apraclonidine
   - Contraindication : Latanoprost ⟹ S/E : uveitis *
2. Chronic cases :
   - Ciliary body atrophy leads to reduced IOP.

Complications and Treatment of Anterior Uveitis 00:53:14

Complications :

1. most common : Complicated cataract
2. Band shaped Keratopathy ✓
3. Cystoid macular edema ✓
4. Pthisis bulbi



Treatment :

1. Steroids (Topical) (Anterior segment)

   +

2. Atropine (mydriatic-Cycloplegic) ✱
   - Cycloplegic : Relax / temporarily paralyse ciliary muscle to relieve pain.
   - mydriatic : Prevent / break formation of posterior synechiae.

Intermediate Uveitis 00:57:08

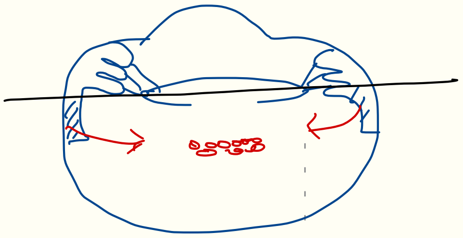
Also known as Pars planitis.

Causes :

1. most common : Idiopathic *
2. Associated with :
   - Sarcoidosis ✓
   - Lyme's disease ✓
   - multiple sclerosis

Active space

Symptoms : Blurring of vision + Floaters ☆ : B/L



Signs :

1. Anterior vitreous cells
2. Snowball opacities ✓
3. Snow banking ✓ (Plaque)
4. Cystoid macular edema : most common cause of vision loss. ☆
5. Periphlebitis *

Treatment :

1. Periocular steroids : Subtenon injection *

↓

Fail

↓

2. Systemic Prednisolone ✓

↓

Fail

↓

3. Immunomodulatory drugs (Azathioprine) *

↓

Fail

↓

4. Pars plana Vitrectomy + Photocoagulation of the Snow banking.

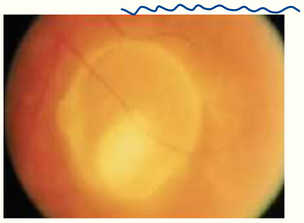
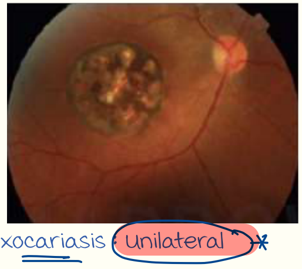
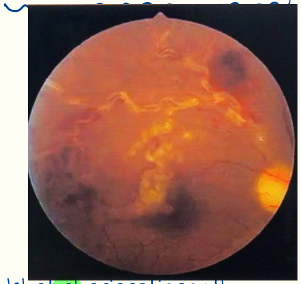
Posterior Uveitis

01:05:46

Inflammation of Choroid and Retina, also known as Chorioretinitis.

C b/c Blood supply : Choroid
outer 4 layers Retina : short post. ciliary. A)

Causes :

Infectious	Non-infectious
1. most common : Toxoplasmosis a. Active : Headlight in fog appearance (due to associated active vitritis)  b. Healed : Punched out scar  2. Toxocariasis : Unilateral * • Leukocoria • Focal necrotising granuloma. 3. Syphilis 4. TB	1. Sarcoidosis • Causes Candle wax dripping appearance (due to perivascular inflammation and sheathing)  2. Birdshot chorioretinopathy • Bilateral • Chronic • most common in females * • Cystoid macular Edema occurs.

C/F : · Floaters
· Painless LOV (though inflamm.)
· Scotoma
Metamorphopsia
Photopsia
→ Retinal Inv.

Rx : Infectious : Treat cause
+
Systemic steroids

Non Infectious : Systemic steroids
or
· Adalimumab : visual studies
· Sirolimus : Sakura trial
☆ · voclosporin : Luminate trial

Panuveitis

01:17:03

Causes :

1. Vogt-Koyanagi-Harada (VKH) Syndrome
    - It affects :

SOME

Ocular	Ears	Skin	Meninges
Bilateral, Granulomatous panuveitis.	Tinnitus	Alopecia	meningismus
Sunset glow fundus.		Vitiligo	CSF pleocytosis
Suiguira sign : Perilimbal Vitiligo.			
⇒ Exudative Retinal detachment.			

2. Sympathetic ophthalmitis
    - Ocular clinical feature : Granulomatous * panuveitis
    - Cause : Accidental penetrating trauma affecting the ciliary body. *
    - Pathogenesis : Trauma to one eye (Exciting eye)

      Ag sensitisation ↓ >2 weeks

      Sympathetic ophthalmitis in other non-traumatic eye (Sympathising eye)
    - Prevention of Sympathetic ophthalmitis in non-traumatic eye :

      * Enucleation of traumatic eye within 14 days. *

☆

Q) Enucleation
- done in cases of sympathetic opthalmitis – T | F

(ENucleation = preventive)

Gyrate atrophy

01:30:55

Gyrate atrophy :

mnemonic : GO AIR

Gyrate atrophy

Ornithine aminotransferase (gene mutation) : Increase levels of Ornithine.

Arginine restriction In diet (Ornithine synthesised from Arginine) *

Active space

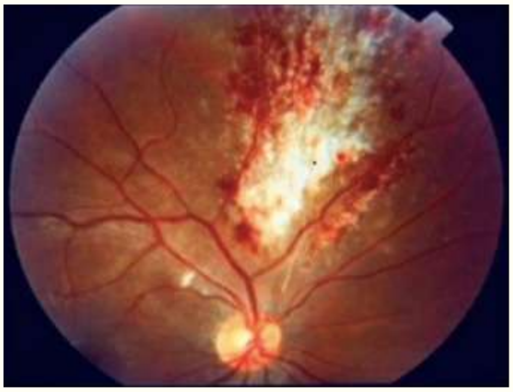
Autosomal Recessive

Ocular HIV:

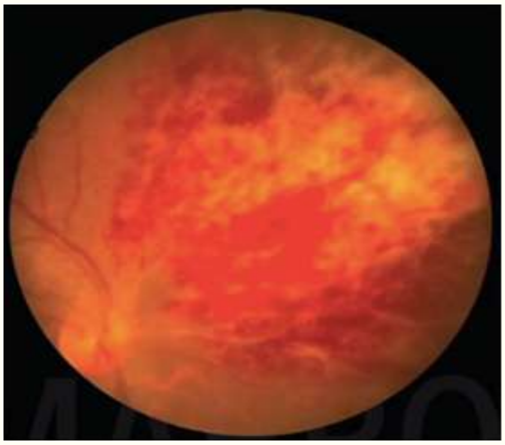
most common ocular manifestation : Retinopathy ✓
most common earliest sign : Cotton wool spots ✓
most common associated tumor : Kaposi sarcoma ✓
most common cause of visual morbidity : CMV retinitis. ✗

CMV retinitis presents with :

1. Pizza pie / Scrambled egg and sauce appearance



2. Brushfire appearance



most common fungal infection: Cryptococcosis.

Active space

Telegram--@latestpgnotes

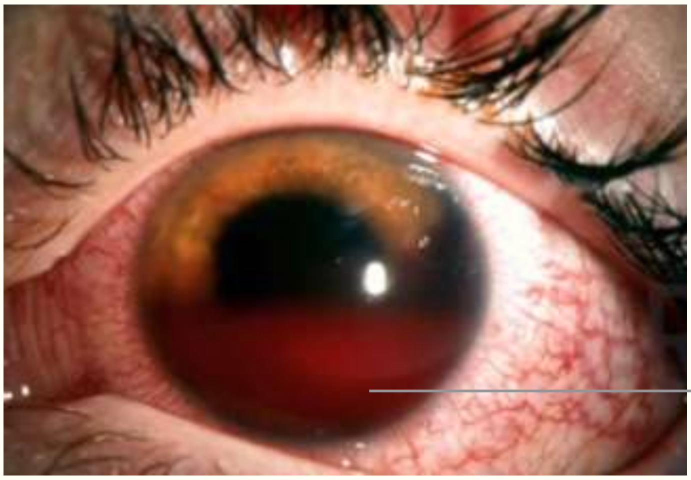
OCULAR TRAUMA

Blunt ocular trauma: Contusion - Causes and Signs

00:01:07

- Causes :
  - Injury with tennis ball.
  - Injury with champagne cork.
  - Fist injury (direct)

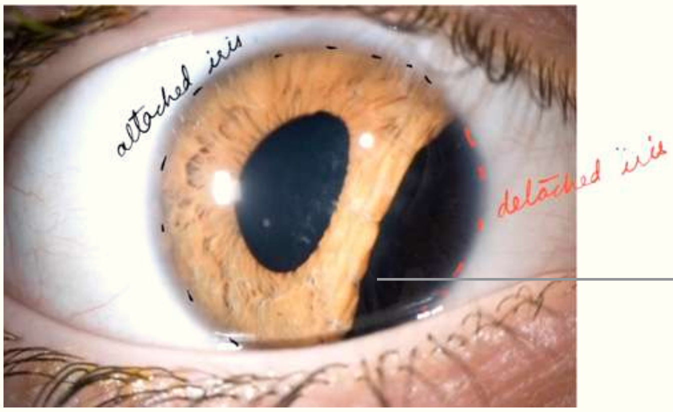
- Signs :
  1. most common is hyphema (collection of blood in anterior-chamber).
     ↳ major Arterial Circle of Iris



* 8 Ball hyphema
↓
Complete AC filled
c̄ black coloured blood

→ Hyphema

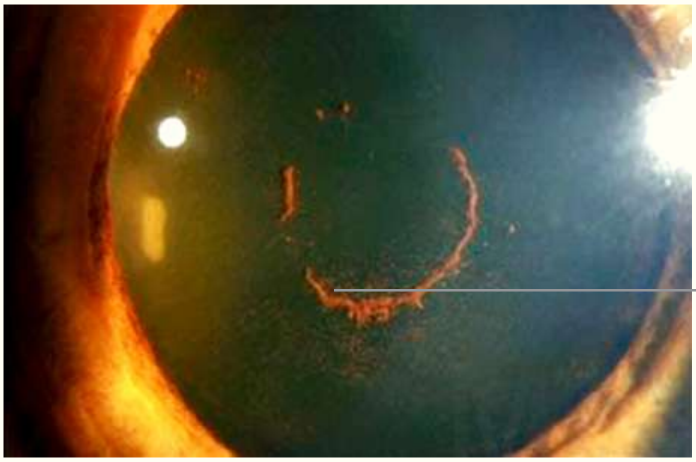
  2. Iridodonesis – tremulous iris.
  3. Iridodialysis – detachment of root of iris from ciliary body *



Blunt Trauma
↓
Iridodialysis leading to
↓
→ D-shaped pupil

Other signs of blunt ocular trauma 00:08:47

4. Rosette shaped cataract (posterior subcapsular cataract).
5. Phacodonesis (tremulous lens). ✓
6. Ectopia lentis (subluxation of lens). ✓
7. Vossius ring (imprint of mitotic pupil on the anterior surface of lens).



8. Commotio retinae – Gray white retina due to edema.
9. Vitreous haemorrhage. ✓ - m/c/c young Adults
10. Traumatic optic neuropathy – RAPD. ✓
11. Choroidal rupture
12. Rhegmatogenous retinal detachment.
13. Glaucoma :
    a. Ghost cell glaucoma – occurs if blood from vitreous haemorrhage seeps into anterior chamber and blocks trabecular meshwork. ✓
    b. Angle recession glaucoma – due to rupture of ciliary body *

2° OAG ←

If edema affects macula ⇒ Berlin's edema
↓
Cherry Red spot

Intraocular foreign body 00:17:40

- Most common intraocular foreign body – iron or steel chips.
- Iron foreign body – MRI is contraindicated.

↓

CT scan is investigation of choice

↓

Leads to siderosis bulbi *

↓

a. Deposition of a rust ring on anterior capsule of lens.

b. Hyperpigmentation of Iris

c. Secondary open angle glaucoma. ✓

↓

Diagnosis – Prussian blue reaction.

Orbital trauma

00:20:40

intra ocular

- ⇒ Ophthalmia nodosa – caterpillar hair causing uveitis. most common fracture in orbital trauma is floor of orbit known as blow out fracture of orbit.

Clinical features:

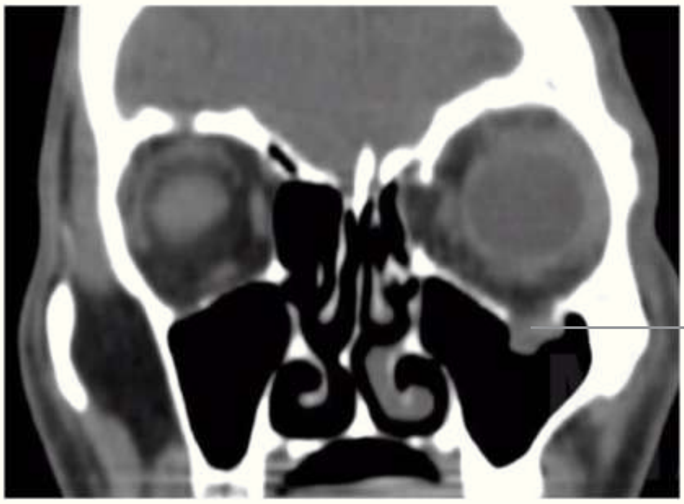
a. X-ray – tear drop sign.

b. Infraorbital nerve anaesthesia.

* c. Double diplopia (diplopia in up-gaze and down gaze, due to muscle entrapment of inferior oblique * and inferior rectus * by fractured bone pieces).

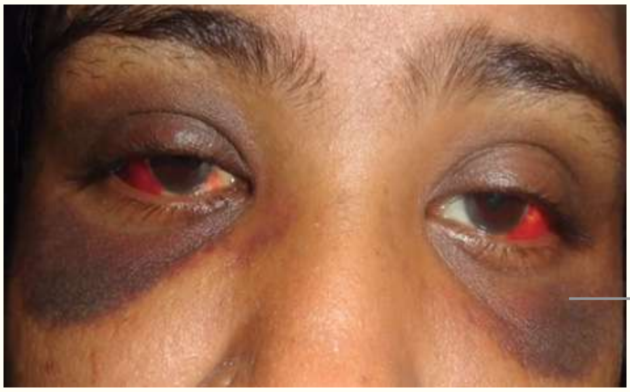
IO
IR

Restrictive squint



→ Tear drop sign

- Fracture of roof of orbit – presents with racoon eyes or, panda eyes.



→ Racoon/Panda eyes

Active space

Chemical injuries

00:26:51

Alkali

- Necrosis
  ↓
  Penetrate t/s

Acid

- Coagulation
  ↓
  donot penetrate

Dua's class
Roper-hall class → Ocular surface burns Grading

Mx : Copious Irrigation of eye : Balanced salt sol^{n}
or
RL
by double eversion of eye

↓

- Topical Abs
- Atropine
- Lubricating eye drops
- Topical sodium citrate drops
  ↓
  Prevent proteolytic activity
- Topical vit-C

Active space

Telegram--@latestpgnotes

EYELIDS

Eyelids or cover of the eye, are movable fold of skin and muscle that can be closed over the eyeball or opened at will.

Anatomy of eyelids

00:00:47

- 3 muscles :

muscle	Nerve supply	Action	On Inability to perform its action/ lesion
1. LPS muscle	3rd cranial nerve	Elevation of upper eyelid	Ptosis
2. muller's muscle	Sympathetic supply	Elevation of upper eyelid	Ptosis
3. Orbicularis oculi	7th cranial nerve	Eyelid closure	lagophthalmos

- 3 glands :

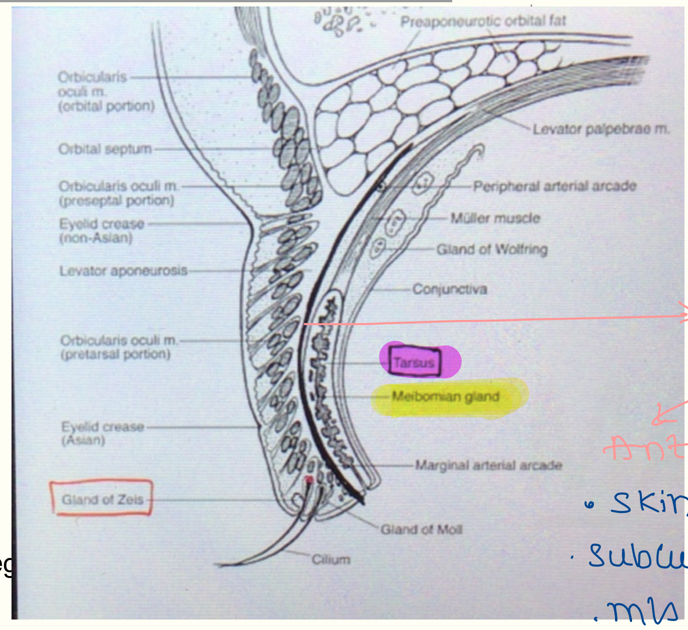
1. Meibomian gland * (tarsal gland)	modified sebaceous gland	Opens posterior to anterior margin of eyelid
2. Gland of Zeis	modified sebaceous gland	Opens at base of eyelashes
3. Gland of Moll	modified sweat gland	Joins the ducts of gland of Zeis *

Pathologiesof eyelids

00:00:47

1. Chalazion.
2. External hordeolum.
3. Internal hordeolum.
4. Trichiasis.
5. Entropion.
6. Ectropion.
7. Distichiasis.
8. Madarosis.
9. Tylosis.

Teleg


Preaponeurotic orbital fat
Orbicularis oculi m. (orbital portion)
Levator palpebrae m.
Orbital septum
Peripheral arterial arcade
Orbicularis oculi m. (preseptal portion)
Müller muscle
Eyelid crease (non-Asian)
Gland of Wolfring
Levator aponeurosis
Conjunctiva
Orbicularis oculi m. (pretarsal portion)
Tarsus
Meibomian gland
Eyelid crease (Asian)
Marginal arterial arcade
Gland of Zeis
Gland of Moll
Cilium

split eyelid
- along Grey line

↑

Grey line

Ant	Post
• Skin • subcut • m/s	• Tarsal Plate

↓

- fibrous t/s
- makes eyelid thick, firm

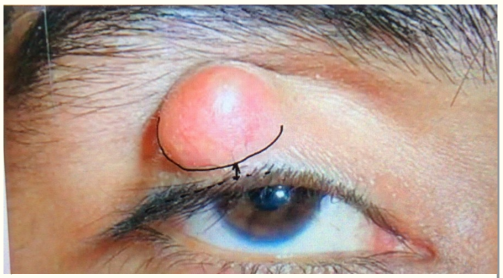
- Conjunctiva

10. Ankyloblepharon.
11. Congenital ptosis.

CML

Chalazion :

- It is lipogranulomatous inflammation of meibomian gland. *
- Non infective.
- Treatment– → posterior part , vertical
  - a) Incision and drainage using chalazion scoop. *
  - ⇒ b) Intralesional triamcinilone. → shrinks the chalazion
- Clinical features – painless swelling away from lid margin.

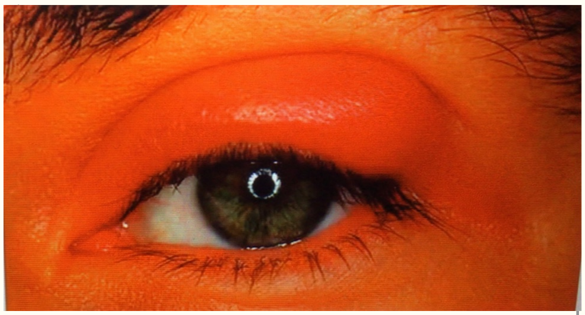


⇒ If reccurent
↓
suspect Sebaceous cell carcinoma

Chalazion : swelling away from lid margin

External hordeolum : Stye

- It is suppurative inflammation of gland of Zeis. *
- Infective agent – staph. Aureus. *
- Treatment –
  - a) Hot compresses.
  - b) Oral antibiotics (if recurrent). *
- Clinical feature – painful swelling at the lid margin.



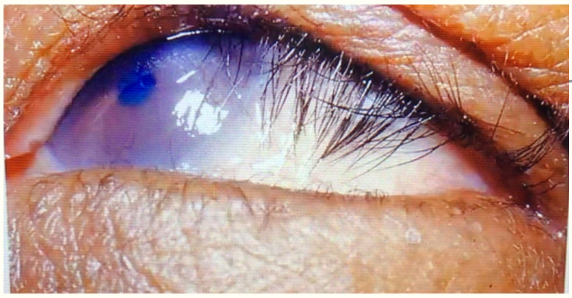
External hordeolum : diffuse swelling at lid margin

Internal hordeolum :

- It is suppurative inflammation of meibomian gland.

Trichiasis :

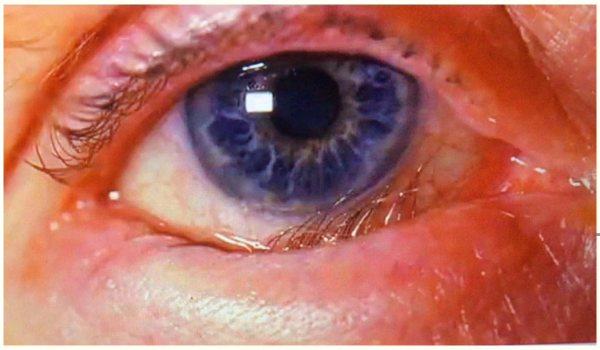
- It's the inward misdirection of eyelashes.
- Treatment –
  a) Epilation.
  b) Cryotherapy of lash base.



Eyelashes turned inwards

Entropion :

- It's the inward turning of eyelid margin.
- Treatment –
  a) Spastic entropion – botulinum toxin injection. ✓
  b) Senile entropion – modified wheeler operation or, weiss operation.
  c) Cicatrical entropion – modified Burrow's operation or, wedge resection.



Eyelids turned inwards

Ectropion :

- It's the outward turning of eyelid margin.
- most common cause is senility.
- Treatment –
  a) medial conjunctivoplasty.

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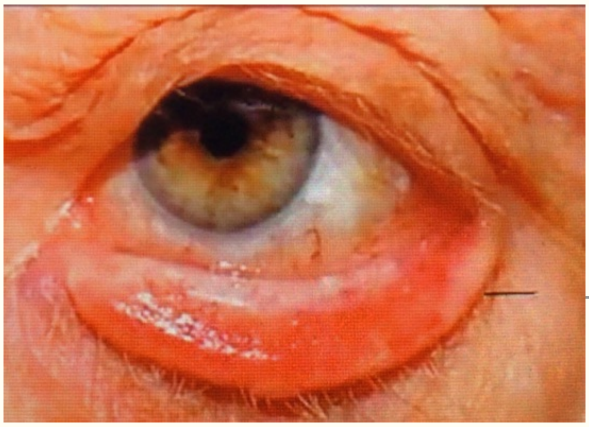
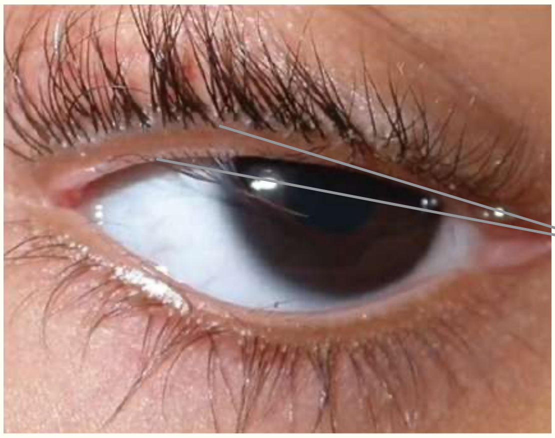
b) Byron Smith operation.



Lower lid turned outward

Distichiasis :

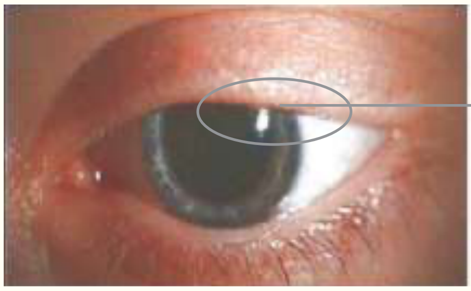
- Double rowsof eyelashes or, extra posterior row of eyelashes.



Anterior and posterior rows of eyelashes

Madarosis :

- Loss of lateral 1/3rd of eyelashes. *



Lateral 1/3rd eyelashes absent

Tylosis :

- It's the thickening of the eyelid margin. *

Ankyloblepharon :

- It's the fusion of eyelid margin of upper lid and lower lid.

Congenital ptosis :

- Occurs due to defective development of LPS muscle.
- Diagnostically : upper lid crease is absent. *

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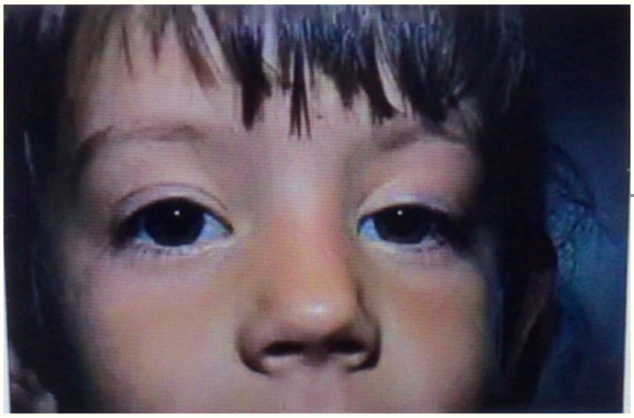
- Lid lag is seen on downgaze.

* eyelid : Neither goes up
Nor goes down



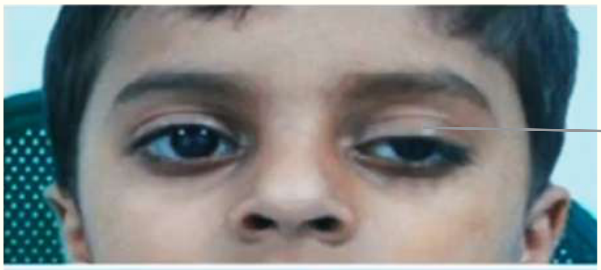
- Treatment-

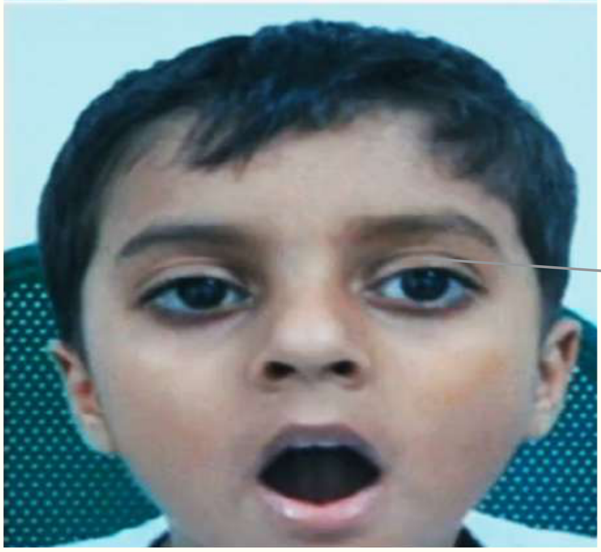
a. mod ptosis + fair LPS fn : LPS Resection

neet ☆ b - Sev. " + poor " " : Frontalis sling Sx

Synkinetic phenomena : Marcus Gunn jaw winking Sx

- d/t Trigeminal & Occulomotor N/v Synkinesis





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COMMUNITY OPHTHALMOLOGY

NPCBVI 00:00:35

National Programme for Control of Blindness and Visual Impairment

- Previously called as NPCB.

Definition of Blindness

- Presenting visual acuity <3/60 in better eye

  Or

- Visual Field limitation to <10 degree

  Same as definition by WHO

Prevalence: 1.05% *

Main Causes :

1. Cataract(62.6% cases) – most common
2. Refractive errors (19.7% cases)
3. Glaucoma (5.8% cases)

C
R
G

Vision 2020 00:04:35

Aim :

1. To reduce blind people to 25 million by 2020
2. To eliminate avoidable blindness by 2020

Launched : In 1999 in Geneva but India adopted in 2001 at Goa.

Disease Covered in India :

1. Cataract ✓
2. Refractive errors ✓

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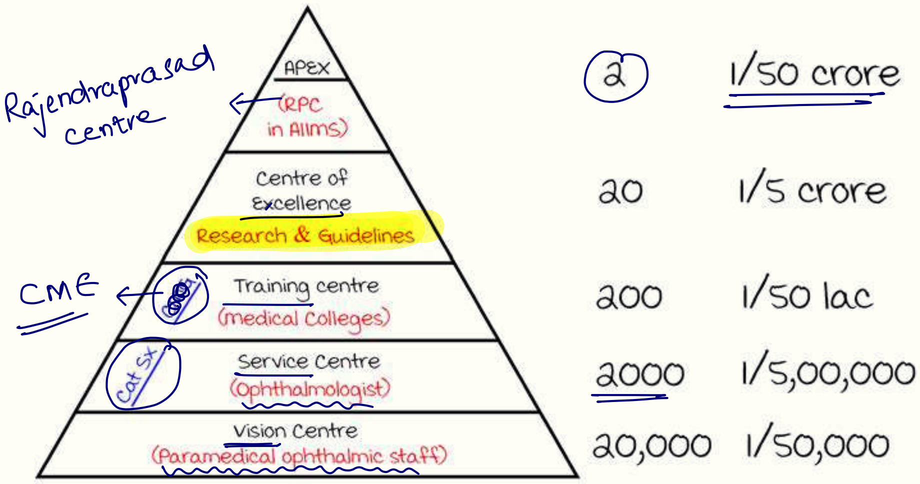
3. Glaucoma : Last disease covered under Vision 2020.
4. Corneal Blindness ✓
5. Childhood Blindness ✓
6. Trachoma
7. Diabetic Retinopathy

Disease Covered Globally :

1. Cataract
2. Refractive errors
3. Childhood Blindness
4. Trachoma
5. Onchocerciasis ☆



School Eye Screening Programme

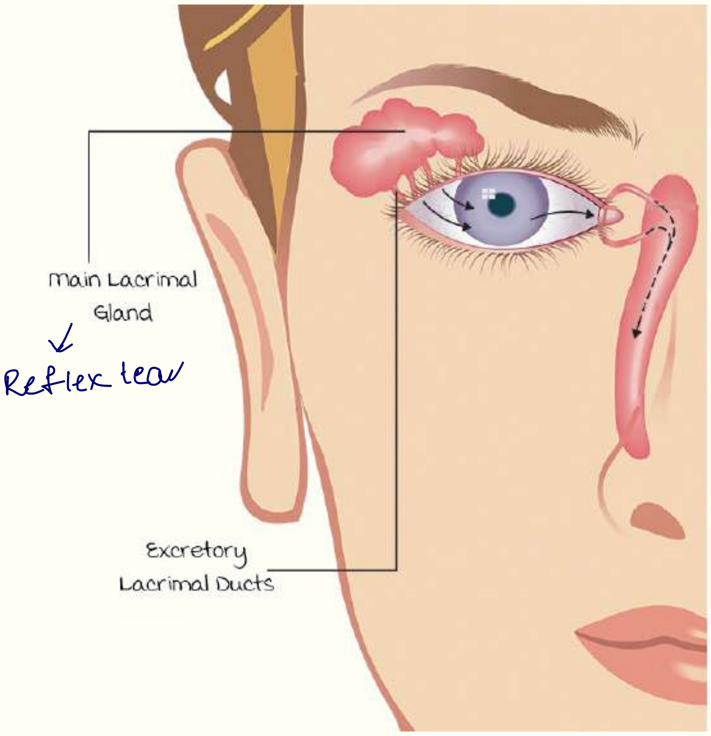
00:11:50

- Done by Teachers
- AIM:10-14 years (Standard 5 to 8) *
- 1 Teacher / 150 Students ✓
- Refer to PHC if vision is <6/9 *

LACRIMAL APPARATUS AND DRY EYE

Lacrimal Apparatus

00:00:33



A. Main Lacrimal Gland

- Situated Superotemporal corner of the eye
- Responsible for Reflex secretion
- Divided into two parts by levator palpebrae superioris
  - Upper Part: Orbital Part
  - Lower Part: Palpebral Part(all the ducts attached) ✱

B. Accessory Lacrimal Glands

- Krause and Wolfring
- Situated in Fornix
- Responsible for basal secretions

What is the parasympathetic (secretomotor) supply of the lacrimal gland

Superior salivatory Nucleus (pons)

↓

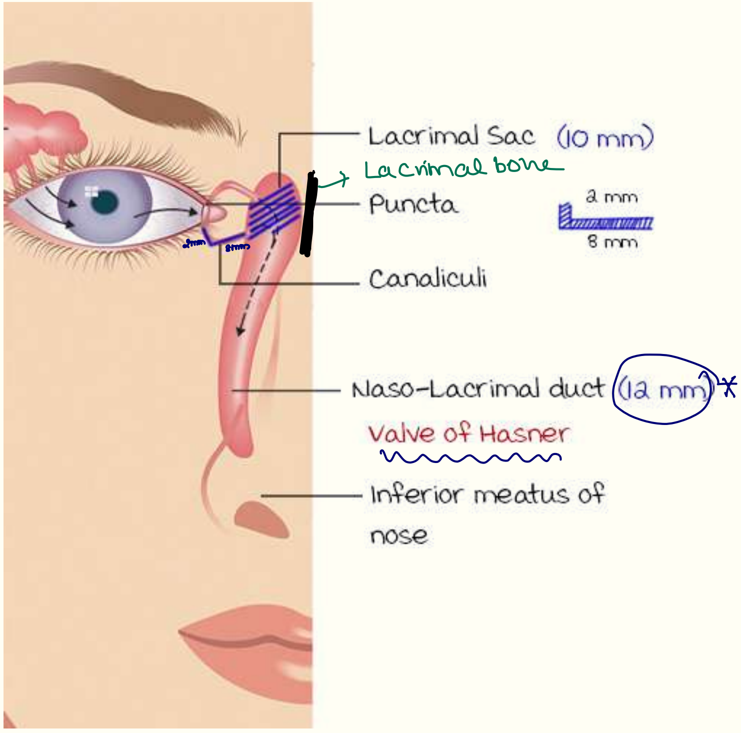
Facial N/v & Greater superf petrosal N/v

↓

Pterygopalatine ganglion

↓

Zygomatic & lacrimal (opthal. division of Trigeminal N/v)



* Lacrimal sac: seperated from middle meatus by lacrimal bone
↓
Removed in DCR ico NLD blockage

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow masterclasses

Watering Eye

00:09:44

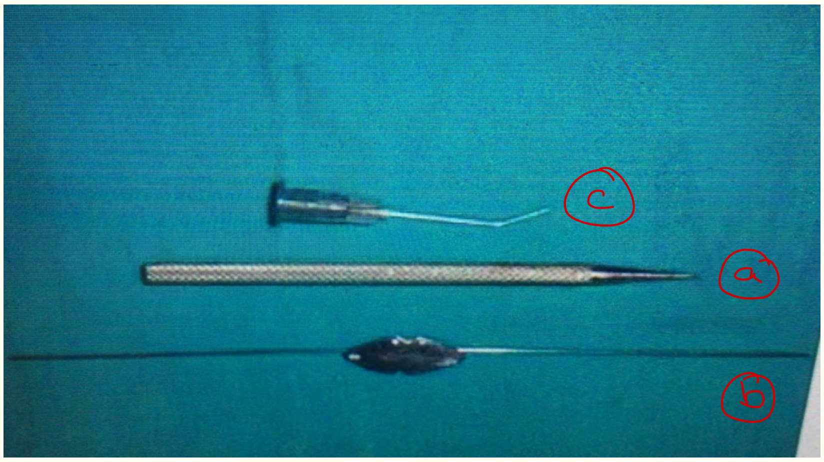
Watery Eye

Hyper lacrimation : d/t emotional
Stress
Or ocular inflammation

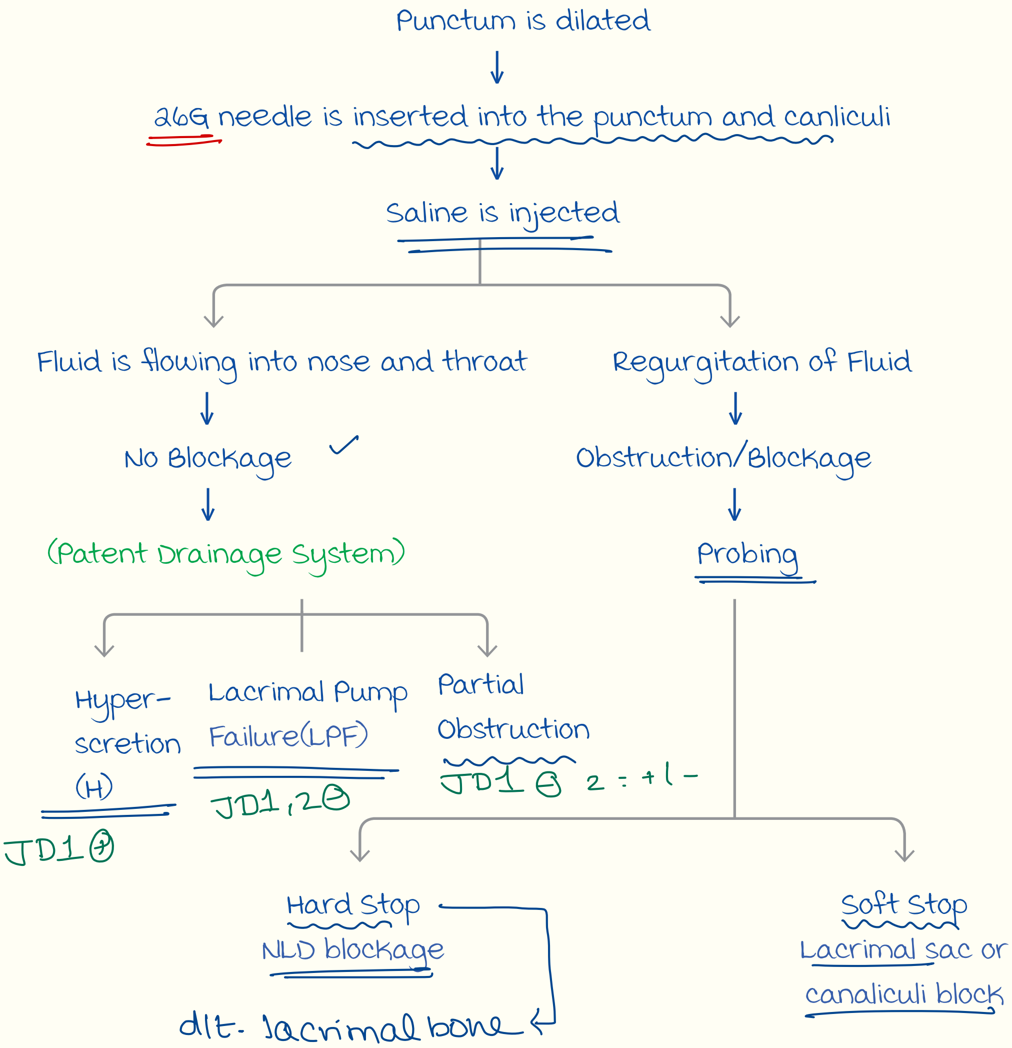
Due to blockage : Epiphora

Testing of watering eye : Done by a Syringing, Probing, and Jones dye test.

a) Nettleship Punctum Dilator
b) Bowman's lacrimal Probe :
c) 26 G Bent needle cannula



Procedure:



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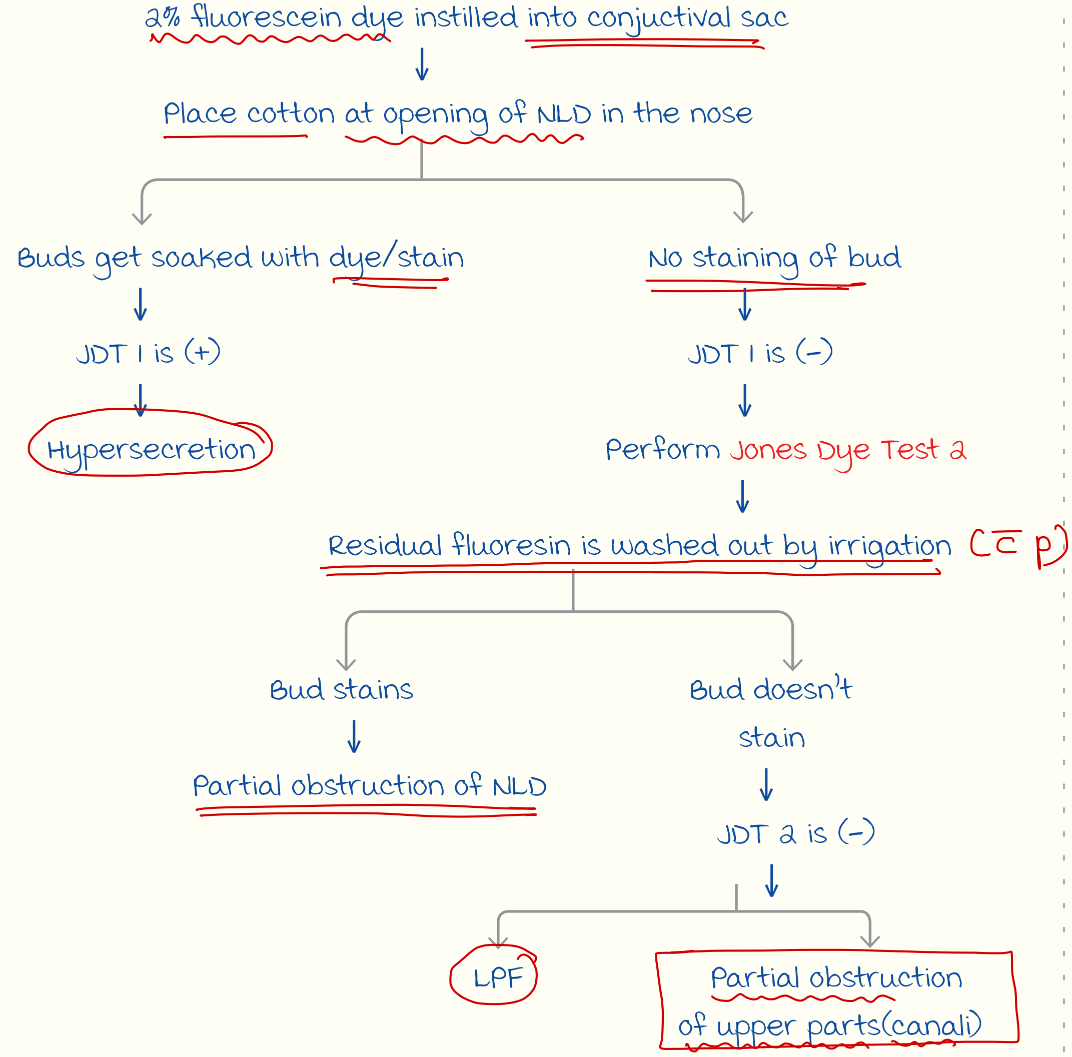
Differentiation between Hypersecretion, Lacrimal pump failure and Partial obstruction

00:17:06

To differentiate between (H), (LPF), and (PO).

Jones Dye Test I (JDT I) :



2% fluorescein dye instilled into conjuctival sac
↓
Place cotton at opening of NLD in the nose

- Buds get soaked with dye/stain
  ↓
  JDT I is (+)
  ↓
  Hypersecretion
- No staining of bud
  ↓
  JDT I is (−)
  ↓
  Perform Jones Dye Test 2
  ↓
  Residual fluoresin is washed out by irrigation ($C\bar{c}$ p)
  - Bud stains
    ↓
    Partial obstruction of NLD
  - Bud doesn't stain
    ↓
    JDT 2 is (−)
    ↓
    - LPF
    - Partial obstruction of upper parts(canali)

- Dacryocystography is used to differentiate LPF and Partial obstruction of upper parts.

Active space

Dacryocystitis

00:23:35

Inflammation of the lacrimal sac.

Congenital

- Complain of watering since birth
- ⇒ Cause: non canalization of NLD(occlusion most commonly at valve of Hasner)
- Treatment:
  1. Lacrimal sac massage+ Topical Antibiotics
  2. If 9-12 month: Syringing ✓ S
  3. If > 1year: Probing ✓ P
  4. If > 4years: DCR ✓ D

Adult

- Cause: Streptococcus Hemolyticus ✗
- Treatment:
  1. Antibiotics(oral) ✓
  2. DCR- if fistula formed

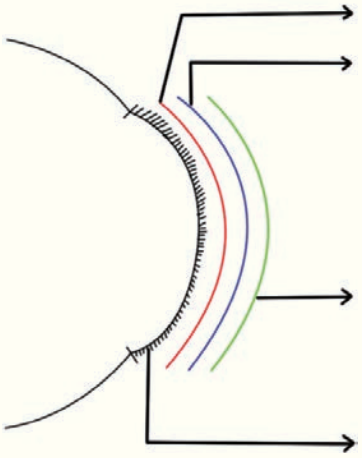
Tumors of Lacrimal Gland

00:27:45

1. Two types:
   - Pleomorphic Adenoma- Benign Tumour
   - ⇒ Adenoid Cystic carcinoma- Malignant Tumour
2. The eye is pushed down and medially.

Tear Film and Dry eye

00:29:00



Adhesion ↑

Innermost layer: Mucin (Goblet cells)

middle layer: Aqueous

Outermost layer: Lipids (meibomean gland)
↳ to prevent evaporation

Corneal epithelium: microvilli

Active space

Dry eye

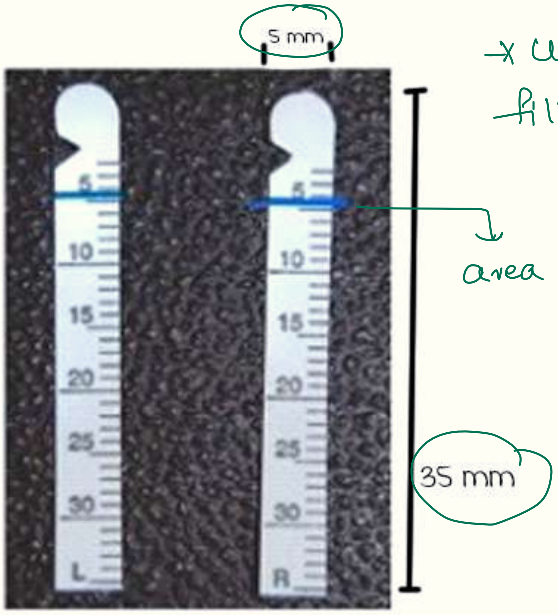
Aqueous Deficiency
Keratoconjunctivitis Sicca (KCS)
Example : Sjogrens Syndrome. ✓

Lipid Deficiency
Evaporative dry eye
Examples :
✓• meibomian gland d/s
✓• contact lens wear

Mucin Deficiency
Examples : Vitamin A deficiency.

Tests :

1. Schirmer test : Quantitative

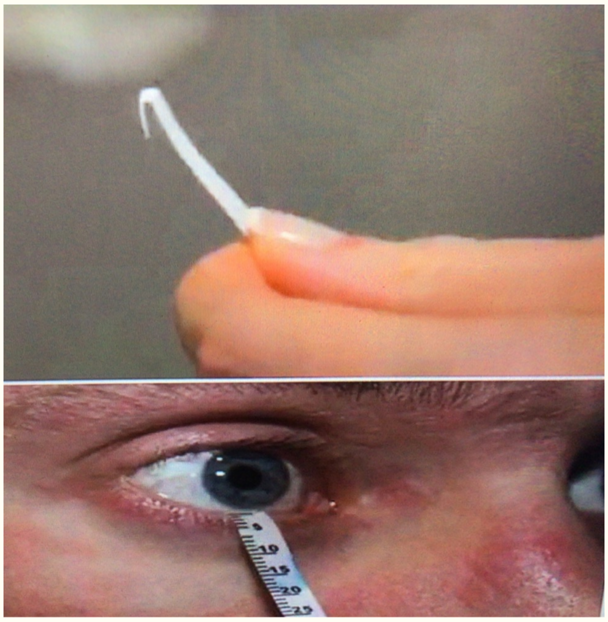


→ uses whatman's filterpaper No 41

area of folding :

strip Folded
↓
inserted into fornix
↓ 5min
See how much strip is wet

- >15mm wet strip → Ⓝ
- <10mm → Dry eye



2. Tear film Breakup time.

2% fluorescein in conj. sac

↓

Illuminated blue light

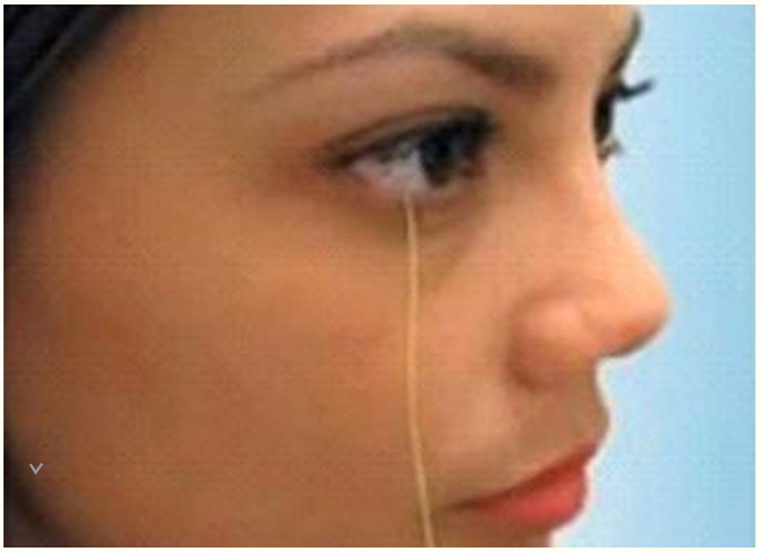
↓

Interval between a Blink and appearance of Ist spot of dryness on cornea (dye : won't be visible)

If <10 sec- Mucin Deficiency

Dry spot at the same location- Corneal disease

3. Phenol Red Thread Test.



Use a thread impregnated with Phenol Red dye (yellow in color) which on contact with tears becomes red.

After 15 seconds

If <6mm - Dry eye

If >15mm - Normal

Active space

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